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FOUNDED BY JAMES PLEASANT PARKER

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XXXIX.

THE DIAGNOSIS AND SURGICAL TREATMENT OF TRIGEMINAL NEURALGIA.*

BY ALFRED W. ADSON, M. D.,
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Trigeminal neuralgia is a disease which is characterized by darting, stabbing and lancinating pains of the face; it affects males and females alike, and is most common during the fourth, fifth and sixth decades. The seat of the disease is the Gasserian ganglion, but its etiology and pathology are still undetermined. The symptoms are manifested by intermittent attacks of paroxysmal pain radiating over one or more branches of the trigeminal nerve, each attack increasing in severity and duration until the pain is more or less constant. The present accepted forms of treatment consist of alcoholic injection of the trigeminal branches for palliative relief, and division of the posterior sensory root of the Gasserian ganglion for permanent relief. The latter, a major operation, once regarded as a hazardous procedure with uncertain results, has been perfected by various surgeons until today it is performed with safety and followed by cure. The incidence of postoperative

*Read before the Seventh Congress of the International Society of Surgery, Rome, Italy, April 7-10, 1926.

complications has been reduced gradually until it has become practically negligible. It is important that a correct diagnosis be established and that the major operation be applied only to cases of true trigeminal neuralgia, since it is of no value in the treatment of atypical neuralgia of the face.

ETIOLOGY.

The specific etiology of trigeminal neuralgia is still unknown. Sir Victor Horsley once attributed the disease to an ascending neuritis following dental caries, but this conclusion has not been accepted since the combination of sensory and motor disturbances of the nerve involved does not exist; however, paresthesia is occasionally found. Not infrequently articles are published by otolaryngologists or dentists, attributing the disease to a specific infection of a sinus, tonsil or tooth, and recording cure on removal of such foci of infection. However, further observation of these patients reveals a recurrence of the neuralgic attacks and demonstrates that the supposed cure was merely coincidental with cessation of that particular attack.

There has been considerable variance of opinion concerning the portion of the trigeminal nerve affected, for example, whether the disease was in one of the branches, in the Gasserian ganglion, or in the sensory root, but since it has been proved that pain radiates apparently over the face, even though the peripheral branches have been divided or avulsed distal to the ganglion, it is evident that the disease must affect some portion of the nerve proximal to the point of division. Abbe suggested that the peripheral branches be divided intracranially just as they leave the Gasserian ganglion, but this is followed in a short time by recurrence of the pain. Injection of alcohol into the nerve or avulsion of the peripheral branches afforded temporary relief, due to temporary cessation of superficial stimuli entering the ganglion or to temporary interruption of stimuli in the ganglion cells. It was then observed that division or avulsion of the sensory root from the pons produced immediate numbness over the area supplied by trigeminal branches, with complete loss of superficial, tactile pain and temperature sensations; however, deep muscle sense was preserved. It was further observed that trigeminal pains were

relieved immediately by this procedure and that they failed to recur if all of the sensory fibers posterior to the ganglion had been divided. Injection of some fixative into the Gasserian ganglion produced a similar change with resultant numbness and relief from pain, but, according to Härtel, trophic keratitis developed in a high percentage of the cases with subsequent loss of the eye. On the contrary, following division of the sensory root, the changes were not so pronounced, and observation of the cornea revealed only microscopic vacuolization without ulceration, unless the cornea had become abraded or infected from the entrance of foreign bodies. Corneal and conjunctival injections were prone to occur if the ophthalmic portion of the Gasserian ganglion was unduly traumatized or manipulated during the operation. Bernard called attention to a fact which he observed in his experimental work on dogs, that trophic keratitis developed invariably following division of the ophthalmic branch peripheral to the ganglion, but rarely if the ophthalmic branch was left undisturbed in a division of the sensory root. He explained this on the supposition that probable sympathetic innervation from the carotid sheath plays a part in the trophic innervation through its distribution to the Gasserian ganglion and the ophthalmic branch.

Frazier, in discussing the etiology and pathology of trigeminal neuralgia, said he believed the disease to be due to sclerosis of the Gasserian ganglion.[15] Dana has ascribed the probable cause to degenerative changes in the ganglion, while Patrick believes that the changes found in the Gasserian ganglion are not sufficient to make a definite statement with regard to the pathology.[28] I have observed that trigeminal neuralgia is most common after the third decade of life, and that it is frequently associated with general arteriosclerosis, cerebro-arteriosclerosis, and occasionally with multiple sclerosis in conjunction with other neuralgic pains, such as intercostal neuralgia, sciatic neuralgia, and atypical neuralgic pains of the face (probably due to sphenopalatine neuralgia). In view of these observations I am convinced that the etiology must be somewhat similar to the etiology of these various diseases, and since the pain is not relieved by removal of the foci of infection, it seems logical to assume that the lesion at the time the neuralgic pain de-

velops is not acute, even though one suspects it to be due to infection, and must, therefore, be due to some sclerotic lesion which has developed within the Gasserian ganglion and represents a postinfectious result.[3]

SYMPTOMS.

The paroxysms of pain incident to trigeminal neuralgia are intermittent and usually remain so unless all three branches are involved, when they become more or less continuous, since, while the pain in one branch may have ceased, it persists in one or both of the other branches. At the onset, the attacks are mild and frequently simulate toothache or shooting pains in the face. The patient often seeks relief at the hands of his dentist, and disappointment follows when the extraction of one or more teeth does not relieve the pain. A physician is then called, and the patient usually receives a varied course of treatment, including the extraction of more teeth, drainage of sinuses, scraping of the jawbone, violet ray, diathermy, and so forth, all without avail. The physician's attention should have been aroused immediately by the sharp, stabbing pain produced by ordinary external irritation over the area supplied by one or more branches of the trigeminal nerve. Washing the face, cleansing the teeth, chewing, swallowing, talking, and even exposure to air currents, are aggravating agents in the production of pain, all of which should aid in making a diagnosis. The attacks occur at first during the day, and not at night unless the patient awakens. The pain differs from that caused by infected teeth or sinuses; in such cases it remains more or less constant regardless of sleep, and generally prevents sleep. The pain of trigeminal neuralgia is described as sudden, severe, sharp, shooting, lancinating and excruciating. Some patients compare it to an electric shock through the face, and others to a red-hot poker jabbed into the cheek or jaws. The pain is of short duration, lasting from a few seconds to a minute or two; and while it lasts, the face and eye may become congested, tears overflow, and patients go into all sorts of contortions in an effort to obtain relief, such as chewing, rubbing the face, stooping forward, and pressing the cheek.

While paresthesia exists in the face, certain areas are more sensitive than others, and these have been referred to as "trig-

ger zones." A trigger zone need not be associated with the particular branch of the nerve affected; the pains may occur over the distribution of the third branch with a trigger zone in the upper lip, or about the ala nasi, in the distribution of the branches of the second division. I observed one patient with double neuralgia in whom the trigger zone was on the opposite side of the face. Superficial injection of the skin has proved of little value in the treatment of trigeminal neuralgia, apart from diminishing the severity and the frequency of the attack; as a rule, it does not afford such complete relief as deep alcoholic injection.

DIFFERENTIAL DIAGNOSIS.

Since the success of treatment depends on the accuracy of the diagnosis, it is well to bear in mind other lesions that produce facial pain. Migraine occasionally produces radiating pain over the forehead and into the eyeball and orbit, but since trigeminal neuralgia is not associated with headache, the history of such periodic headaches with radiating pain into the ophthalmic branch of the nerve rules out the possibility of true trigeminal neuralgia. Pain incidental to sinus infection is more or less constant, rather than paroxysmal, and is frequently associated with local tenderness; the pain occurs independent of external irritation and is not aggravated by washing the face, talking, chewing, or swallowing, as is the case in trigeminal neuralgia. "Jumping" toothache occasionally simulates trigeminal neuralgia, and, at the onset, may be difficult to differentiate from trigeminal pain, but, if the pain persists, pathologic evidence will soon be forthcoming, either by careful physical or X-ray examination. Then, too, with dental caries the pain usually persists after the patient retires, while with trigeminal neuralgia the pain, at the onset, is rarely initiated during the patient's sleep.

Glossopharyngeal neuralgia simulates trigeminal neuralgia in being intermittent and paroxysmal; it is brought on by similar causative factors, and occurs at about the same time of life, except that it follows the distribution of the glossopharyngeal nerve instead of that of the trigeminal nerve. The trigger zone in cases of glossopharyngeal neuralgia is situated in

the tonsillar fossa and the paroxysms are initiated by yawning, swallowing, talking, laughing, and coughing, with needle-like pains radiating to the ear drum instead of down the branches of the fifth nerve. If doubt exists with regard to the differential diagnosis, thorough cocainization of the throat is advisable; this anesthetizes the trigger zone and causes a cessation of glossopharyngeal pain. If the lesion is of trigeminal origin, the paroxysms of pain will not be influenced.

Sphenopalatine neuralgia, as described by Sluder, is "a lower half headache," and is characterized by more or less constant, boring pain, situated apparently in the superior maxilla. It is sometimes most pronounced under the malar bone, and at other times in the gums of the superior maxilla and the hard palate, with radiations toward the sphenoid and down the neck. Sluder reports relief in many of these cases from injections of alcohol and phenol into the sphenopalatine ganglion, and the external application to the ganglion of silver nitrate. Other surgeons have reported less favorable results, which coincide with my experiences. Besides the boring, pressing pain, there may be burning, smarting, or prickling pain in the same area, and occasional involvement of the eyeball and even of the area supplied by the third trigeminal branch of the nerve, causing unpleasant sensations in the lower lip and the margin of the tongue. This pain differs from that of trigeminal neuralgia in that it is more or less constant; it is not paroxysmal, and is moderately severe, but not sufficiently so to incapacitate the patient, unless he is of a neurotic temperament, in which case the pain is always greatly accentuated and incapacitating. Neither deep alcohol injection into the branches of the trigeminal nerve nor division of the sensory root affords relief in the treatment of this type of neuralgia; as a matter of fact, the condition is aggravated, since the numbness, instead of affording relief, produces additional irritation.

The foregoing syndrome, which Sluder describes, may exist independently or may be associated with trigeminal neuralgia. If the patient possesses a sturdy nervous system, he will complain but little or not at all of the unpleasant sensations persisting after operation, and relief from the severe trigeminal pain; if, on the other hand, he is hypersensitive and neurotic,

he will be very likely to forget the relief afforded him by the division of the sensory root and complain of the burning sensations, disregarding practically the existence of the former neuralgia. For this reason it behooves the surgeon to familiarize himself thoroughly with all of the complaints incidental to the facial pain before he advises any therapeutic measure for relief.

TREATMENT BY ALCOHOL.

The injection of alcohol in the treatment of trifacial neuralgia was first described in 1902, and by Schlösser in 1903. Härtel, in 1914, described the technic for injecting the Gasserian ganglion with alcohol, and since then numerous articles have appeared on the subject. The technic that has been generally followed was described by Levy and Baudouin in 1906, and slightly modified by Patrick in 1907.[27] Treatment by alcohol is indicated when the diagnosis is not clear, since if the condition is a true trifacial neuralgia, temporary relief will be obtained. It is also of value if patients are poor operative risks, or if they are being prepared for the radical operation. The results are only palliative; the average interval of relief afforded is nine months, and, as repeated injections are administered, the interval becomes shorter. It is true, that in a few instances relief has been afforded for three or four years. Two of the patients under my observation were relieved for twelve years, but these are exceptions to the rule.

Patrick describes the injection of alcohol as follows: "The needle is about 8 cm. in length and 1.5 mm. in diameter, and is of the trocar and cannula type, the cannula being graduated in centimeters in order to indicate the depth reached. Before inserting the needle the skin is cocainized; as soon as the proper depth has been reached, the trocar is replaced by a glass syringe, fitted into the cannula, and about 2 c. c. of 80 per cent alcohol solution is injected. The patient usually experiences pain when the nerve is approached, but this is quickly followed by numbness. Since the injection under local anesthesia is more or less painful, Doctor New, of the Section on Oral and Plastic Surgery of the Mayo Clinic, employs the technic suggested by Frazier; for example, instead of using local an-

esthesia, he anesthetizes the patient partially with nitrous oxid, until he passes into the analgesic state; this relieves him of the experience of pain when the needle is inserted. If, upon entering the nerve, he complains of considerable discomfort, a deeper anesthesia is induced prior to administration of the alcoholic injections, thus minimizing the pain and the shock incident to the injection. The mandibular branch is injected at its exit from the foramen ovale and is reached by placing the needle through the skin at a point 2.5 cm. in front of the descending root of the zygoma which almost coincides with the anterior bony border of the external auditory meatus. The needle is directed slightly upward along the base of the skull and a little backward at a depth of 4 cm. The superior maxillary division is injected by placing the needle through the skin, 0.5 cm. posterior to the external angle of the orbit underneath the zygoma, directing it vertically to the anteroposterior line but inclined slightly upward in a direction which should attain the nerve, at a depth of 5 cm. at its emergence from the foramen rotundum."

TREATMENT BY SURGERY.

Primarily operation on the fifth nerve for trigeminal neuralgia was confined to simple section of the branch affected, then to the removal of sections of the branch, when Thiersch is supposed to have removed the root and branch of the division involved. Mears, in 1884, suggested the removal of the Gasserian ganglion for the relief of trigeminal neuralgia. In 1886, at the suggestion of Ferrier, Horsley and MacEwen attempted operative interference in cases of trigeminal neuralgia.[22] The ganglion was approached through the temporal bone, and the operative procedure was limited to a single division of the sensory root. Because of the high mortality, however, the method was abandoned. Sir William Rose, in 1890, after excising the superior maxilla and trephining the base of the skull, with the foramen ovale as a center, curetted the ganglion. Hutchinson, in 1898, suggested partial resection of the ganglion and sensory root with a view to preserving sensation in the cornea. Cushing, in 1900, published a paper on the extirpation of the Gasserian ganglion, and again in 1905 and 1906 reported series of cases. Hartley, in 1891,

opened the skull laterally, following the osteoplastic flap method devised by Wagener, lifted the dura and removed the ganglion, with a successful result. He reported this case in 1892. A few months later, Krause published an article describing the same method with records of four cases. Because of the complications and the mortality associated with ganglionectomy, Spiller suggested division of the sensory root (physiologic extirpation) in lieu of ganglionectomy; it had been discovered that the spinal sensory roots, once severed proximal to the ganglion, do not regenerate, as was proved by Frazier and Spiller in 1900 and 1904, and by Van Gehuchten in 1903. The operation suggested by Spiller was performed by Frazier in 1900, and proved to be successful. De Beule, in 1913, recited his attempts to develop retrogasserian neurotomy in the treatment of obstinate facial neuritis in 1897, and in succeeding years, until it was officially recognized in 1908. In this operation there is the slight difference that the approach, instead of being in the middle fossa, follows the ridge of the petrous bone. Since the development of the operation of division of the sensory root, in preference to ganglionectomy, numerous refinements have served to minimize the surgical hazard, such as the control of bleeding, the prevention of unnecessary trauma to the ganglion, accurate and complete division of the sensory root, and preservation of the motor root. In 1919, Frazier found that it was possible to preserve the motor root; Cushing undertook the preservation of the motor root at about the same time. Kanavel and Davis, in 1922, discussed the anatomy of the trigeminal nerve with reference to preservation of the motor root and the probable cause of facial paralysis from injury to the superficial petrosal nerve and the geniculate ganglion of the facial nerve, which may explain the occasional occurrence of facial paralysis.

In March, 1922, I discovered accidentally that the motor root could be exposed readily from the mesial side by gentle traction and elevation of the sensory root and the ophthalmic portion of the ganglion.[2] I took occasion to publish the details of the technic, since it differed from methods previously described, and because it was possible to save the root in every case. Since that time I have been able to avoid damaging the motor root in 193 consecutive cases. Since the publication of

the article by Kanavel and Davis on the surgical anatomy of the trigeminal nerve, I have taken special care to prevent elevation of the arachnoid sheath underneath the ganglion and sensory root, and have thus avoided injury to the superficial petrosal nerve. As a result I have performed the operation in 71 consecutive cases without facial paralysis ensuing. The exact technic of the procedure is detailed further on.

Frazier, in 1925, reported the results obtained in a series of 25 cases, over a period of ten years, in which he saved the ophthalmic portion of the sensory root without recurrence of pain in the ophthalmic division.[16] In my study of 839 cases of true trigeminal neuralgia, a comprehensive review is made of the branches involved, and it is evident that the ophthalmic division was affected in a large percentage. It is true that the disease in many cases may be confined to the second and third branches for a long period, but in view of the fact that the ophthalmic division is involved in 30 per cent, I have preferred to divide the sensory root completely.

PREPARATION OF PATIENT FOR MAJOR OPERATIONS.

Since the injection of alcohol into the deep branches of the trigeminal nerve affords only palliative relief and, in some instances, no relief at all, and since peripheral avulsion is only palliative without the possibility of repeating the operation, it becomes necessary to seek elsewhere for a measure which will afford permanent relief. To accomplish this, division of the sensory root has been the operative procedure universally accepted. Before advising operation, the surgeon should be positive of his diagnosis of true trigeminal neuralgia and should not attempt to relieve atypical pain in the face by hazardous methods when it is not due to disease of the Gasserian ganglion. It is well not to perform the major operation for mild symptoms of short duration, without previous preliminary injections of alcohol, since these patients are likely to forget the severity of the pain and complain of the numbness and the "woodeny" feeling that follow division of the sensory root. If the patient has previously learned that the paroxysms of pain will recur unless radical measures are taken for its

relief, and that numbness must be accepted as a postoperative result following division of the sensory root, he is only too willing to accept the inconvenience in lieu of the excruciating pain. In every case with a short history of involvement of only one branch, from one to three injections should be given, not only as a therapeutic measure but as an educational course. The disease occurs occasionally in patients beyond the ages of sixty or seventy, and in these particularly it is important that a careful medical examination should be made to determine the possible presence of organic disease. Mild interstitial nephritis, diabetes, arteriosclerosis and multiple sclerosis are not contraindications to operative treatment, but before operation is undertaken careful investigation of the kidney and electrocardiographic studies should be made to determine whether or not the nephritis or myocarditis present is grave enough to contraindicate operation, whether the diabetes is subject to control, or whether the high blood pressure with a history of light apoplectic attacks does not present too grave a risk. A surgeon naturally tends to avoid major operations in the presence of one of these constitutional diseases, but frequently these conditions are found in patients who have been tortured with trigeminal neuralgic pain for from fifteen to twenty years, and, when they no longer obtain relief from repeated injections or attempts at avulsion, beg for radical interference despite the additional surgical hazard they may have to assume. In these instances, the radical operation with the additional risk seems to be the humane course, especially since the mortality is extremely low, considering the average age at which these patients undergo operation. Conservatism should be emphasized, of course, in the management of this group of patients, and if injections of alcohol afford any appreciable amount of relief, the radical operation should be avoided. In some patients of advanced age with marked cardiovascular disease, manifested by dyspnea, edema and attacks of apoplexy, surgical interference is contraindicated, and attempts at relief must be made by repeated injections of alcohol of the peripheral branches or ganglion, or by peripheral avulsion. In the preparation of patients for operation on the ganglion, every possible precaution should be taken, and the preparation should be as careful as for laparotomy.

OPERATIVE TECHNIC.

The operation may be performed under general or local anesthesia; general anesthesia may be induced by ether, ethylene, or nitrous oxid. Dowman and Coughlin are performing the operation with success under local anesthesia. It has been my experience that if the operation is carried along under local anesthesia until the skull has been opened and the dura partially elevated, and a short period of ether anesthesia is then induced, division of the sensory root can be performed with much less suffering to the patient and with greater ease and rapidity. The ether should be discontinued immediately after division of the sensory root, and the wound closed under local anesthesia. The patient is thus permitted to regain consciousness before leaving the operating table. I believe that there is very much less shock associated with this procedure than with prolonged general anesthesia, or with prolonged operation under local anesthesia.

The operative field is prepared, the patient is placed on the operating table on his back with his head in a ganglion headrest, and the table is then adjusted until the zygoma is in a plane parallel with the floor. The headrest is adjustable, so that the head can be tilted towards the surgeon, permitting easy exposure of the middle fossa and at the same time allowing the blood or cerebrospinal fluid to flow away from the Gasserian gangilon or sensory root.[5] The incision is made in front of the ear, extending upward and backward from the zygoma;[25] the lower point of the incision is situated 1 cm. in front of the tragus on the lower margin of the zygoma, and the upper point 5 cm. above the helix of the ear (Figs. 1, 2 and 3). The temporal fascia is also incised for a distance of 5 mm. in each direction at right angles to the incision along the upper margin of the zygoma, before the muscle is incised. The skull is then trephined and the opening enlarged to a diameter of about 3 cm. It is well to enlarge the opening in a downward and mesial direction so that the approach will be directly mesial along the floor of the middle fossa (Fig. 4).

When the dura is being elevated, care should be taken to avoid injuring or tearing it, since this adds to the difficulty

of controlling bleeding of the middle meningeal. It is well to free the dura from the bone at the outset with a dural elevator which is curved on the posterior surface and has sharp edges; this instrument helps to loosen the dura if it is densely adherent to the bone. I have rarely encountered this difficulty with patients under the age of sixty, but with patients in the late sixties or seventies, the dura is usually very adherent and frequently much thinner than in younger persons.

If, by chance, the middle meningeal artery has been torn at this stage or in a subsequent stage of the elevation, bleeding can be controlled by placing the ganglion retractor (which is a curved, flat spatula with a small electric light attached) on the vessel below the point of hemorrhage (Figs. 5 and 6). In proceeding with the elevation of the dura, the surgeon should bear in mind that the floor of the middle fossa almost invariably shows individual variations, and he should not be misled by the various irregularities or grooves that may be present. Having once assured himself that the dissection was started in the middle fossa, he will find the middle meningeal artery and the third branch of the trigeminal nerve passing through the foramen spinosum and the foramen ovale at the innermost and dependent portion of the middle fossa. Should the surgeon discover during the dissection that he is approaching an elevation anteriorly, he should at once direct the dissection downward toward the hollow of the fossa; should he approach an elevation posteriorly, he is again going astray and should direct the dissection back to the most dependent portion of the middle fossa and proceed mesially, following the general direction of the middle meningeal artery, which is being elevated with the dura. Occasionally the middle meningeal artery may take an anterior or posterior direction from the foramen spinosum, and not be observed in the line of dissection. The surgeon will find that, if he is careful to adjust the head so that the zygomatic plane is parallel with the floor, he will be oriented at all times and will not waste time attempting to study anatomic relations in the middle fossa before exposure of the third branch. The dura should be elevated gently with a ganglion retractor and separated from the bone by cotton pledgets held in a bayonet forceps. The pledgets used for this purpose, as suggested by Frazier, are made of dental roll, cut

in various sizes; these rolls come in 6-inch lengths, and after being sterilized are cut into pledgets varying from 5 to 15 mm. in length. They are particularly useful, since they have sufficient body to aid dissection and absorb blood. If it is necessary to control venous oozing, a dental pack with a linen suture attached serves the purpose very well, and permits continuation of the operation.

After the middle meningeal has been exposed, it is convenient to ligate it just as it leaves the foramen spinosum; it is dissected free from the dura at the foramen, an end suture is then passed around the artery with a modified aneurysm needle, and it is tied with the aid of a small knot tier (Fig. 7), similar to the Boettcher knot tier used in tonsillectomy. The knot is placed in the suture outside of the cranial wound, but is pushed down and secured around the artery with the knot tier. Three knots are made: the first two are loose, granny knots, the second knot tightening the first, and the third is reversed in order to secure the first two as a square knot to prevent slipping. The artery is then divided with long narrow scissors distal to the ligature. In case the artery is injured at the foramen spinosum by a sharp spur, hemorrhage might be controlled by a wooden plug, but preferably with Horsley's bone wax. If stiff wax is used, difficulty will be encountered in molding it into the foramen spinosum; soft wax, on the contrary, will be forced out by the pulsations of the middle meningeal artery. If the hard wax is warmed to a temperature of 100° F., and then applied, it can be very easily molded into the foramen and then held in place for a few seconds until it cools. This procedure can be carried out most effectively by the aid of a dental pledget, dipped in sodium chlorid solution of the same temperature, which prevents the cotton from sticking to the wax. The middle meningeal having been controlled, the third branch is exposed just mesial and slightly anterior to the foramen spinosum. It is not necessary to continue the dissection anteriorly to expose the second or first branches, but it should be carried along the outer margin of the third branch and ganglion. The dura should be elevated gently from the lateral portion of the ganglion by a rotating movement of the dental pledgets rather than by direct pressure toward the ganglion, as this will frequently produce rests in the arachnoid.

During the elevation of the dura from the third branch and the lateral portion of the ganglion, a reflection of dura to the bone is encountered lateral to Meckel's fossa; it has been my experience that the procedure will be greatly facilitated if this is divided with a sharp knife posteriorly to the middle portion of the petrous bone in a plane parallel to the zygoma (Fig. 7). It has been observed also that the dissection can be readily continued without injury to the arachnoid and without danger of elevating the ganglion and its sheath from Meckel's fossa which, according to Kanavel and Davis, may produce trauma to the superficial petrosal nerve, and in turn give rise to a disturbance in the geniculate ganglion and occasional facial paralysis of temporary duration. Following this particular technic, I have performed 71 consecutive operations without the occurrence of facial paralysis. It is my hope that the method is responsible for this improvement in results, but further experience will be necessary to establish a causal relation.

After the third branch and the lateral half of the ganglion have been exposed, the dissection is carried posteriorly and upward until pulsations are seen beneath the arachnoid (Fig. 8). These are set up in the cerebrospinal fluid around the sensory root posterior to the ganglion, since the arachnoid is not attached to the sensory motor roots but to the margin of the semilunar ganglion. The dissection should be carried posteriorly to the ridge of the petrous bone, exposing an area of pulsating arachnoid about 1 cm. in diameter. During the dissection, the dura is held taut and retracted laterally and upward with a ganglion retractor to avoid injury to the third, fourth and sixth cranial nerves. The pulsating arachnoid is then incised with a small right angled ganglion knife; a semilunar incision is made from the mesial side to the lateral, with a bow over the ridge of the petrous bone, permitting the arachnoid flap to fall toward the middle fossa, liberating the cerebrospinal fluid, and exposing the sensory root. The motor root lies ventral to the sensory root and usually along the mesial side, but may take a median ventral position until it reaches the margin of the ganglion, when it takes an abrupt turn downward and laterally underneath the Gasserian gang-

lion, into a separate sheath, to become continuous with the third branch.

In order to preserve the motor root I follow the technic which I have previously described,[2] and which I have used in 193 consecutive cases with satisfactory results. Since the sensory root lies superficial to the motor root and spreads out like a fan to the Gasserian ganglion, it can be brought into view very readily by retracting it in a downward, lateral direction, with a small right-angled blunt hook at its junction with the ganglion (Fig. 9). This retraction places the sensory root under tension and relaxes the motor root, which is not elevated with retraction of the sensory root; furthermore, the line of traction is in the direction of the normal course of the motor root.

In about half the cases in which this procedure is followed, the motor root can be seen to form a small bend or elbow on the mesial side of the sensory root; in the other cases it may be slightly adherent to the sensory root. When the sensory root is rotated laterally, the motor root is found to lie in a relaxed position ventral to the sensory fibers, the latter being under tension and the motor fibers in the relaxed position. The motor root is then isolated and held mesially by the blunt hook, while the sensory fibers are either avulsed or cut proximal to the ganglion (Fig. 10). Care should be taken to avoid trauma to the motor root, as it will give rise to temporary paralysis of the masseter, temporal and pterygoid muscles (Fig. 11). It has been observed also that extensive trauma to the ophthalmic portion of the ganglion may result in marked conjunctivitis and trophic keratitis, immediately following operation. Previous to closure of the wound, venous oozing should be controlled either by small muscle pledgets, wax, if the oozing is from the bone, or a small iodoform gauze pack, which should be removed in from twenty-four to forty-eight hours. The wound is closed in the usual manner, preferably with interrupted silk sutures. The advantage of the straight incision is that it provides ample exposure, permits splitting of the temporal muscle, and is quickly made and closed, and does not injure the nerve innervating the frontalis portion of the occipitofrontalis muscle.

The postoperative care is similar to that demanded following any major procedure, except that special care of the eye is necessary. During the operation the lids are held securely closed by a strip of adhesive which is replaced by a Buller's shield when the patient regains consciousness. The shield is worn for three or four days, and is then replaced by a pair of glasses fitted with a corneal shield;[6] a special celluloid cup is attached to the rim of the frame to prevent the entrance of foreign bodies during exposure to dust and wind. It is usually unnecessary to wear the ganglion glasses when in the home or office, but the protection of the eye against the entrance of foreign bodies cannot be urged too strongly, since the cornea, as well as the skin supplied by the branches of the fifth nerve, is anesthetic. I recommend daily irrigation of the eye with 2 per cent boric acid solution, especially after exposure to dust or wind, to insure thorough cleansing and the removal of any small foreign bodies which might have lodged. I have observed that herpes will occasionally appear over the distribution of the affected branches after operation. This is of no particular significance unless it affects the ophthalmic branch, when the eye should be covered with a Buller's shield until the herpetic patches disappear, in about ten days, as a rule. If the herpes occurs in or around the nose or on the lip, a mild medicated ointment is all that is necessary. If it occurs within the mouth an antiseptic mouth wash is efficacious. Occasionally a patient may bite the anesthetized cheek. The teeth should be examined to make sure that there is no unnecessary irritation from bridge work, an irregularly placed tooth or a portion of a decayed one. Following operation, the patient should, as a rule, remain in the hospital for about a week, and be kept under observation for dressings for from three to seven days longer.

REVIEW OF CASES.

This study is based on the records of 839 patients examined and treated at the Mayo Clinic for trigeminal neuralgia; 448 were males and 391 females. The average age on admission was fifty-three years; the youngest was seventeen years, and the oldest eighty-eight. The former was the only patient in the second decade. However, Patrick reported that three of his

patients gave a history of the onset of trigeminal pain at the age of seven. In my series seventeen patients were in the third decade, 71 in the fourth, 169 in the fifth, 259 in the sixth, 225 in the seventh, 87 in the eighth and 10 in the ninth.

The average duration of symptoms, at the time of the patient's admission, was seven and one-half years. A number of these patients had suffered for from ten to twenty years, while others had complained for a short time only. The delay in seeking relief, in the former group, was due, no doubt, to inaccuracy of diagnosis or to the patient's fear of the surgical treatment which at one time was regarded as very hazardous. Occupation has no particular causal relation to the disease; 132 of my patients were either in business or professions; 335 were laborers, and 372 were housewives.

In 261 of the 839 cases there was an important complicating disease; nephritis was present in 17, diabetes in 15, arteriosclerosis in 141, multiple sclerosis in 36, syphilis in 5, migraine in 26, intercostal neuralgia in 6 and sciatica in 20. No doubt, a history of migraine, sciatica and intercostal neuralgia was frequently not recorded during the earlier part of this series.

In eight cases the patient's complaint was referable to the ophthalmic division alone; in 137 to the superior maxillary division alone, in 167 to the mandibular division, in 95 to the ophthalmic and superior maxillary divisions, in 8 to the ophthalmic and mandibular divisions, in 291 to the superior maxillary and mandibular divisions, in 125 to all three branches, and in 12 the division was not recorded. Summarizing, in 253 cases (30 per cent) the disease affected the ophthalmic division alone or in conjunction with the other two divisions; in 439 (52 per cent) the superior maxillary division alone or in conjunction with the other two; in 551 (65 per cent) the mandibular division alone or in conjunction with the other two.

In 541 cases (64 per cent) the right side was affected; in 285 (34 per cent) the left side, and in 13 (2 per cent) both sides. It is interesting to note that the disease occurs almost twice as frequently on the right side; when the neuralgia was bilateral, it appeared simultaneously on both sides. However, the paroxysm was rarely equally severe on both sides at the

same time; that is, the attacks in one branch or on the one side would subside when the paroxysm was at the height on the opposite side. In one case of bilateral neuralgia, the pain appeared on the opposite side seven years after the initial attack.

For convenience of study, I have classified the series into two groups: a medical group, comprising those cases in which treatment consisted of deep alcoholic injection, peripheral avulsion, and so forth, and a surgical group, comprising those cases in which, after the use of palliative measures, radical operation was performed, either ganglionectomy, in the early cases of the series, or division of the sensory root in the later cases.

In the entire series of 839 cases a total number of 2,338 deep injections of alcohol were administered; when it was successful, relief lasted from nine to eighteen months on the average. Some of the injections were given elsewhere, but the majority of them were given in the Mayo Clinic. In approximately 30 per cent injection was incomplete because the nerve could not be located or because scar tissue, which had followed repeated injections, prevented infiltration of the nerve. Two patients obtained relief from injections of alcohol for a period of twelve years. Peripheral avulsion was performed 237 times, and the average duration of relief was seven and three-tenths months. There were 532 other palliative operations, such as sinus drainage, tonsillectomy, extraction of teeth, without any specific relief, making a total of 3,107 palliative procedures for the relief of this disease. The result of this review justifies the following deductions:

1. Deep injection of alcohol is the best palliative procedure, since it can be repeated without serious complications.

2. Peripheral avulsion is not justifiable unless injection of alcohol is ineffectual in cases in which the major operation is either unsuitable or is refused.

3. Operations on the sinuses, tonsils and teeth offer no specific relief, but are not contraindicated if definite infection exists.

The deep injection of alcohol serves a triple purpose: (1) it may be used to establish a diagnosis, provided relief and

anesthesia follow; (2) it affords relief from pain when the general condition of the patient does not permit division of the sensory root, and (3) it is of educational value, since it teaches the patient that relief can be obtained but that he must accept numbness in lieu thereof. I have found it best to advise from one to three injections, or even more, if they are effective, when only one branch is affected. If two or all three branches are involved and there is a long history of pain, an exception to this rule may be necessary. When a single division is affected, and that for a short time and not severely, section of the sensory root is inadvisable, since the patient may complain more of the numbness after operation than he did of the pain, and will fail to realize the potentialities of his pain. On the other hand, it is unfair to ask patients to continue with palliative treatment until relief is no longer afforded them, and the risk of operation has become too serious, since the major operation will enable them to return to their civil and social activities completely relieved from pain, so that they can enjoy life, instead of being forced by their incapacitation for work and enjoyment to be a charge on their relatives and their community.

Peripheral avulsion is rarely, if ever, justified, since it cannot be repeated, there being no twigs at the peripheral foramina after one thorough avulsion, except in the case of the ophthalmic division. This nerve, however, is difficult to inject on account of the danger of producing paralysis of the third, fourth and sixth cranial nerves in the orbit.

Of the 839 cases studied, operation was performed in 587; of these 20 were performed between 1909 and 1916; ganglionectomy was performed in 15 cases, and avulsion of the sensory root in 5. In one case a Hutchinson operation was performed, that is, resection of the ganglion with preservation of the ophthalmic portion. Division of the sensory root was performed in 371 cases, with preservation of the motor root in 193 consecutive cases since 1922. This series included seven cases in which previous operations had been performed on the ganglion or the sensory root; in four the operation had been performed elsewhere; in three cases I had operated previous to 1918. In all seven cases the symptoms appeared in the ophthalmic

or the superior maxillary division owing to extension of the disease to that portion of the ganglion which had not been previously involved and which was still supplied by sensory fibers. These had been missed at the initial operation because of improper illumination of the bloody field. Since the adoption in 1918 of the present technic, which provides thorough exposure of the sensory root with an illuminated ganglion retractor, no cases have recurred. In all cases since 1917, comprehensive sensory examination has been made after operation to determine the extent of anesthesia and the possibility of any fibers having been left undivided, and whether the motor root was functioning. I have found that if a great deal of sponging is done during the operation, there will be diminished function in the muscles supplied by the motor nerve. If no injury occurs to the motor nerve, the temporary impairment due to the sponging will soon disappear, but in any event it behooves the surgeon to be very careful in the dissection.

The complications due to bleeding and poor exposure have been fairly well overcome by employment of the technic described. In the series of 387 surgical cases, conjunctivitis occurred in 34, keratitis in 12 and iritis in one. In 2 of these cases enucleation of the eye became necessary at a later date; in 6, corneal opacity ensued. Ocular palsy involving the sixth nerve occurred in 2 cases, early in the series; cerebral disturbance with postoperative coma occurred in 5. In 2 of these death occurred, 1 from apoplexy, thrombosis of the middle cerebral artery being revealed at necropsy, while in the other only cerebral edema was found. Recovery in the remaining 3 occurred six days after operation, delirium occurring just previous to recovery. I was unable to explain this complication, but in each instance marked cerebral arteriosclerosis was present. Four patients complained of impairment of hearing following operation, and likened their symptoms to the feeling of having water in the ear; in 1 case the tympanum ruptured spontaneously and drained cerebrospinal fluid for three weeks, during which time mild symptoms of meningitis appeared. Mercurochrome and gentian violet were administered intravenously, and may or may not have aided in the complete recovery. In the analysis of this complication it

was observed that, during the decompression, zygomatic cells were opened occasionally and that unless these were plugged immediately with wax, there was a likelihood of cerebrospinal fluid traveling on through the mastoid cells, the antrum and the middle ear. This would account for the sensation of which these patients complained. Nine patients continued to complain of various nervous phenomena, such as the postoperative "woodeny" feeling in the face, or pain in other parts of the face or head. Twenty-one patients complained of unpleasant sensations in the cheek or eye, describing the sensation as of a hair being drawn across the cheek, or as if there were a burning in the cheek or a foreign body in the eye. No doubt these sensations were due to involvement of either the sympathetic nervous system or of the pain fibers associated with deep muscle sense, or possibly to involvement of the sphenopalatine ganglion. Care in making a differential diagnosis previous to operation cannot be emphasized too much; it is essential that the exact character of the pain should be determined and whether or not it is chiefly paroxysmal; the two types exist simultaneously and, if a burning pain of a nonparoxysmal type predominates, the reactive result is bound to be unsatisfactory. Paralysis of the face is a complicating symptom, since it predisposes to keratitis and conjunctivitis. Complete paralysis occurred in 4 cases immediately following operation; recovery took place in all. In 24 cases partial paralysis of the seventh nerve occurred from three to seven days after operation, varying in degree; in most cases it was very slight, with perhaps only a suggestion of difficulty in closing the lid or moderate drooping of the face. Not infrequently recovery occurred before the patient's dismissal from the hospital.

Some of these complications occurred during the early development of the operative technic, and it has been possible to reduce their frequency greatly. Some of the deaths might have been avoided if the sad plight of these patients had not stimulated me to operate when, after peripheral avulsion and repeated injection, they failed to gain further relief from palliative measures. One's better judgment is at times overruled by sympathy. Since many patients with complicating diseases survive the operation and are relieved of their severe pain,

the hazards should be explained very plainly to the patient and relatives before operation is undertaken, and if they choose to assume the risk the patient should be given the chance of the major operation.

One patient, aged 61 years, died from hemorrhage; 3 (69, 37 and 66 years of age) died from meningitis. The patient 37 years of age was suffering from osteomyelitis of the jaw at the time of operation. Three (72, 68 and 39 years of age) died from pneumonia; 2 (65 and 67 years of age) from cerebral complications as previously mentioned (1 from apoplexy and the other from cerebral hemorrhage); 2 (62 and 60 years of age) died from pulmonary embolism; 2 (both 67 years of age) from cardiac failure; 1 (aged 67 years) from diabetes and bronchopneumonia, and 1 (aged 66 years) from cerebroarterial sclerosis with nephritis.

SUMMARY.

Trigeminal neuralgia is a major disease which fails to disappear spontaneously, but continues with increasing severity until it incapacitates the patient and makes of him a permanent invalid, forcing him to withdraw from his social and civil activities and become a charge upon others.

Before treatment is instituted, the patient should be thoroughly acquainted with the progress of the disease and the numbness which necessarily follows alcoholic injection or division of the sensory root, and which must be accepted in lieu of the excruciating pain incident to the disease.

Injections of alcohol and peripheral avulsion afford temporary relief, but division of the sensory root confers instant and permanent relief.

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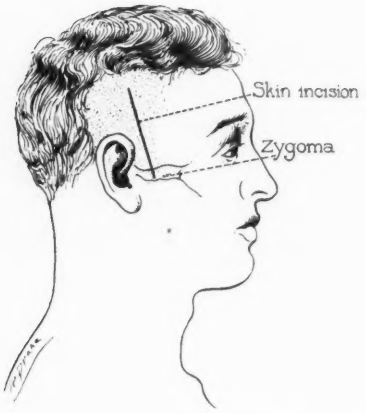

Fig. 1. Temporal incision for division of the sensory root of the Gasserian ganglion.

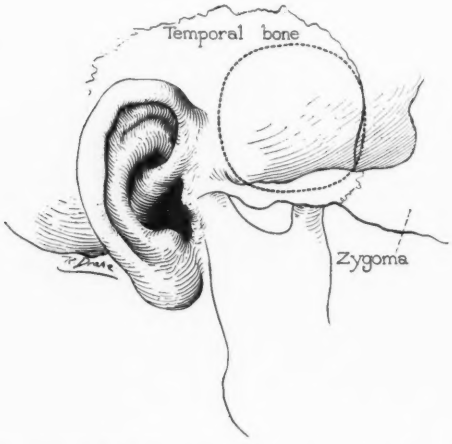

Fig. 2. The dotted line illustrates the position and the size of the temporal decompression employed in division of the sensory root.

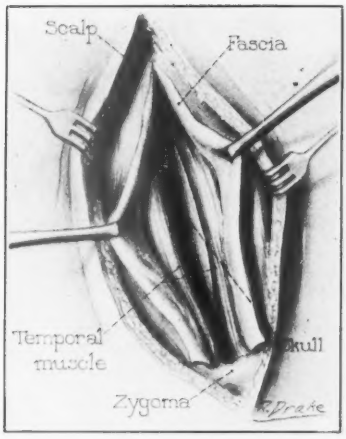

Fig. 3. Incision of the tissues in the temporal area.

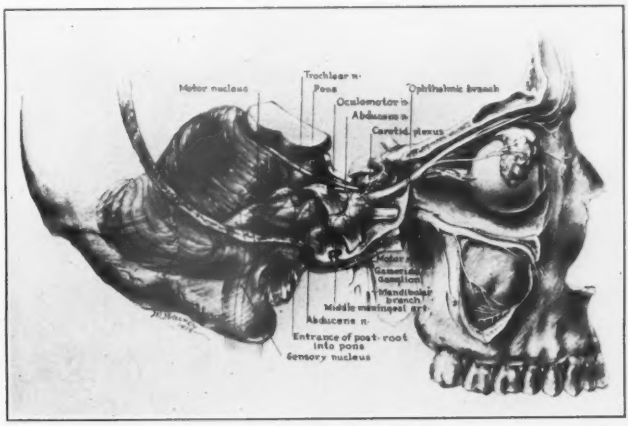

Fig. 4. Illustrating the relation of the ganglion to its surrounding structures, as well as the communications between the carotid plexus and the ophthalmic portion of the ganglion.

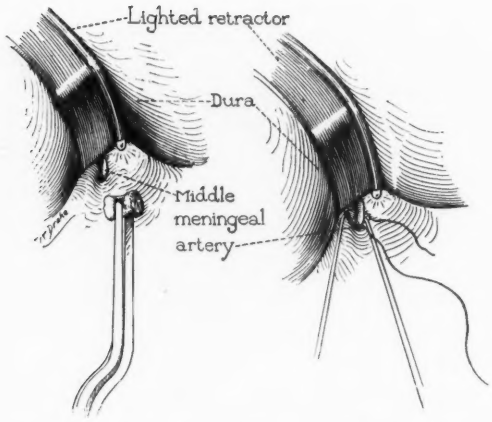

Fig. 5. Elevation of the dura with the ganglion retractor, demonstrating the dental pledget and the bayonet forceps used in the dissection; also, the first step in ligating the middle meningeal artery.

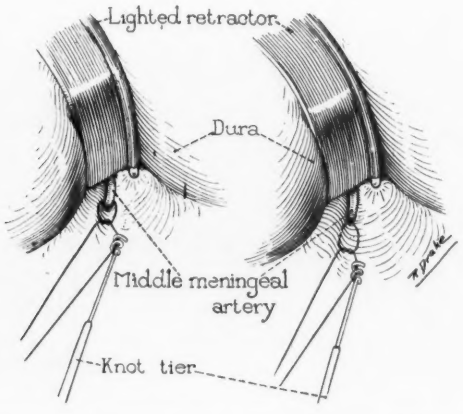

Fig. 6. Illustrating the method of exposing and ligating the middle meningeal artery with the aid of the knot tier.

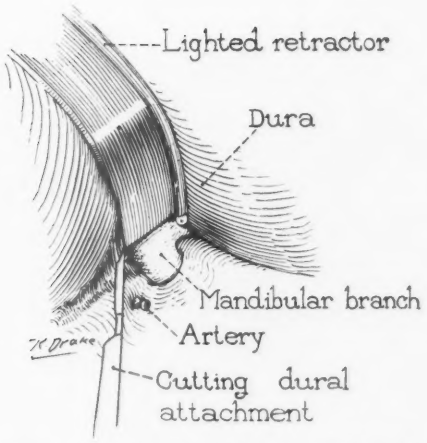

Fig. 7. Exposure of the ligated middle meningeal of the third branch of the Gasserian ganglion, and of division of the dural reflection to the temporal bone in order to prevent elevation of the ganglion or of its posterior sheath, thus avoiding injury to the superficial petrosal nerve which lies underneath the ganglion.

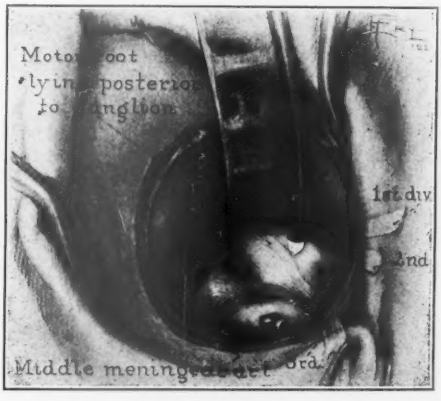

Fig. 8. Exposure of the Gasserian ganglion with its arachnoid covering previous to posterior division of the sensory root.

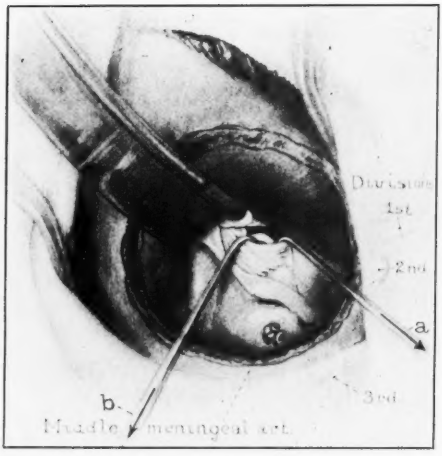

Fig. 9. Following incision of the arachnoid, hooks *a* and *b* are placed in order to elevate the sensory root and retract it downward and forward, placing the sensory root fibers and the ganglion on tension, but relaxing the motor root.

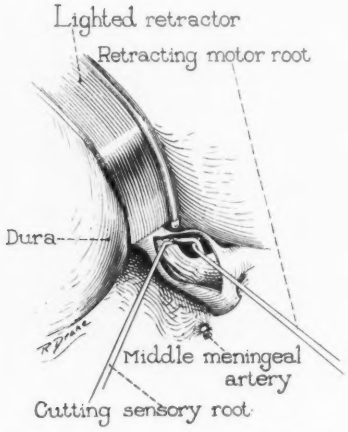

Fig. 10. Isolation of the motor root on the mesial side of the sensory root, with division of the sensory root with a sharp right angled knife.

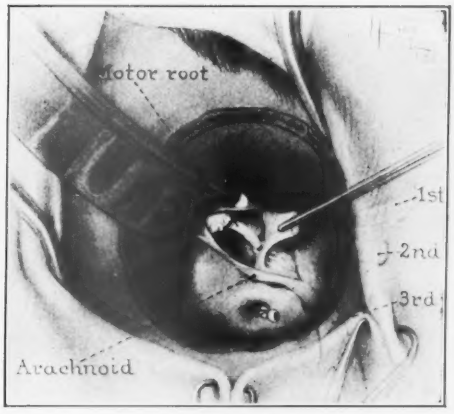

Fig. 11. Elevation of the divided posterior sensory root fibers, exposing the motor root as independent fasciculus entering a separate sheath beneath the ganglion before submerging into the third branch of the trigeminal nerve.

XL.

MONAURAL TESTS ON BONE ACUITY UNDER VARIOUS CONDITIONS, WITH SOME GENERAL COMMENTS ON BONE ACUITY.

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and

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Two papers have already been published on quantitative tests for air and bone acuity under varying conditions in the external auditory canals. The first paper showed the effects of plus and minus pressures of the air in the external auditory canals on air and bone acuity. The effect of complete occlusion on bone acuity was also considered. The second paper took up the influence of complete occlusion on air acuity and also considered the effect of both partial and complete occlusion on bone acuity.

It was found during the course of these experiments that the point of application of the bone activating telephone influenced the bone acuity. It was therefore determined to establish quantitatively the variation in monaural acuity dependent on the position of the bone activating receiver and to further modify the condition of the external auditory canal by repeating the water filling experiments described by Wheatstone. The technic used in the investigation is described in the first paper and need not be considered here.

Wheatstone discovered in 1827 that filling the external auditory canal with water produced an effect on bone transmitted sound similar to that occasioned by occlusion of the external

meatus. In both instances a lateralization to the affected side was noted. It is obvious that the mechanical conditions under which the drum membrane operates are entirely different when it is flanked by water on one side than when it is flanked by air on both sides. If, therefore, exact measurements of the bone acuity are made under the two conditions, any difference between the two might furnish a clue to the mechanics of the actual reinforcement for bone transmitted sound caused by occlusion of the external auditory canal.

Six observers were used in this test. The head of the observer was placed on one side and supported in such a way that the lower external canal was entirely free from any obstruction. The canal of the upper ear was gradually filled with warm water by means of a pipette, and care was taken to avoid any possible trapping of air. In Fig. 1, the distances of the letters from the base line 1 represent the benefits to bone transmission by filling one ear with water. Tests were made at the five frequencies shown. The different letters refer to the six persons tested, there being two letters for each condition at each frequency corresponding to the two ears of a person. The readings on the right ear filled with water are underlined. Omissions occur where the maximum energy available was not sufficient at some of the frequencies for some of the individuals. The points show the ratio of necessary energy for hearing with the external canal filled to the necessary energy with the canal open. Points at ratios less than unity mean that filling the ear with water was detrimental to bone transmission. The majority of points, however, are at ratios greater than 1, showing the water filling was a benefit to bone transmission. Transmission was tried from three locations on the head: (I) from the forehead; (II) from the mastoid adjacent to the ear which was filled with water; and (III) from the mastoid on the opposite side from the ear which was filled with water.

An examination of the data shows that the benefit due to the presence of water in the external auditory canal depends on all three factors given as variables: i. e., the individual, the frequency and the point of test. For instance, in testing from the forehead at 256 p. p. s., observer b was more benefited by the water in the canal than were any of the other observers. At 512 p. p. s., however, observer b was the least bene-

fited. This did not hold true, however, when the tests were made from the mastoids.

One general conclusion which can be drawn from the data is that on the average the benefit obtained by filling the external canal with water is less at the higher frequencies of 1024 and 2048 p. p. s. than at the lower frequencies. This was true for all positions of test. At 2048 the average change is less than unity, indicating a detriment due to the water. The benefit increases as the frequency decreases down to 256 p. p. s., and for the tests from the mastoid on the side of the filled ear, the average benefit at 128 p. p. s. is slightly greater than for 256 p. p. s. Testing from the forehead, the average benefit is about a factor of 10 in intensity for the three lower frequencies used, and is somewhat greater than this for tests from the mastoids. Averaging all observers and all the frequencies used for each position of test, it is found that the benefit is about a factor of 4 from the forehead; 10 from the mastoid on the side of the filled ear; and 6 from the mastoid on the opposite side from the filled ear.

In Fig. 2 are shown the relative normal sensitivities to bone transmission of the individuals under normal conditions. Their sensitivities are plotted on the same system of intensity ratio as is used in Fig. 1, and in such a way that the larger this ratio the greater is the sensitivity. The tests were made from three positions, (I) the forehead, (II) the right mastoid, and (III) the left mastoid. It is to be noted that the energy actually supplied by the bone transmitter at the various frequencies is not known and the values given in Fig. 2 are therefore not comparable as between frequencies. At any given frequency, however, the sensitivities of the different individuals and also the sensitivity of any one individual, as tested from the three positions given, may be compared. Similarly Fig. 3 shows the relative sensitivities of the six individuals to air transmitted sounds at the five frequencies used, those of the right ears being underlined.

A consideration of the results allows the following conclusions to be drawn:

1. With the ears open, bone transmission is better from the mastoids than from the forehead, not only on the average but

also individually, with but few exceptions. The difference averages an intensity factor of about 2.2 at 128 p. p. s., 7.6 at 256, 10 at 512, 1.4 at 1024, and 15 at 2048 p. p. s. The advantage of bone transmission from the mastoids over that from the forehead holds with one ear filled with water as well as with both ears open.

2. Bone transmission from the mastoids is, on the average, also more affected by water in the ear than is bone transmission from the forehead.

3. Good bone transmission is slightly more closely associated with good air acuity than with poor air acuity. This is in agreement with the conclusions reached in previous papers, but is in opposition to what is often stated as to the relation between bone and air acuity in pathologic cases.

4. For the lower frequencies, the filling of the ear with water effects the greatest improvement in the ears which are poorest in bone transmission.

5. Filling the external canal with water causes a greater improvement in bone transmission from the mastoid on the side which is filled than from the mastoid on the other side.

In order to separate the effect of the water pressure on the drum membrane from the effect of the occlusion of the canal by the water, the experiments were repeated with occlusion with dental impression compound, which shuts off the canal without any direct mechanical interference with the drum membrane. These tests were made on the same individuals as were used in the water filling tests.

The improvement in the bone transmission due to the wax occlusion is shown in Figs. 4 and 5. The improvement due to occlusion of one ear is shown in Fig. 4 on the same basis and scale as that used in Fig. 1 for water occlusion, the letters representing the individuals tested and the crosses showing the averages of the other points. A given letter represents the same individual in Fig. 4 as in Fig. 1. It was not possible to fill both ears with water in a way to have this the only factor involved, but it is possible to occlude both ears with wax, and the resulting improvement is shown in Fig. 5. In order to indicate the improvements in bone acuity a little more clearly, the average values from Figs. 1, 4 and 5 are given in Fig. 6.

Although small differences are not decisive, a comparison of these curves allows several conclusions to be drawn. These may be grouped under three headings.

A. EFFECT OF THE POSITION OF THE BONE ACTIVATING RECEIVER.

1. Bone acuity from the forehead is more affected than from the mastoids through occlusion of the external auditory canals with wax. This is true for occlusion of one or both canals.

2. Bone acuity from the mastoid is more affected than from the forehead through filling the external auditory canal with water. This is the reverse of the result with wax occlusion.

3. Occlusion of one external auditory canal affects the bone acuity from the mastoid on the closed side more than from the mastoid of the opposite side. Exceptions to this are found at the highest frequency with water occlusion and at the two lower frequencies with wax occlusion. At least one of these, the wax occlusion at 128 p. p. s., seems to be a real exception to the generalization, as may be seen from a consideration of the separate observations in Fig. 4, which show that at 128 p. p. s. none of the six individuals tested support the above generalization.

B. EFFECT OF THE TYPE OF OCCLUSION.

1. Wax is distinctly more effective than water in improving the bone acuity from the forehead.

2. Wax in both ears is a little more effective than wax in only one ear in improving the bone acuity from the forehead and from the mastoids.

3. With one ear occluded, the bone acuity from the mastoid on that side is slightly more affected by water at the lower frequencies and by wax at the higher frequencies.

4. With one ear occluded, the bone acuity from the mastoid opposite the closed ear is in general more improved by wax occlusion than by water occlusion.

C. EFFECT OF THE FREQUENCY OF THE TONE.

1. In general the maximum effect of occlusion is at 256 p. p. s. and decreases markedly as the frequency rises until at 2048 the water occlusion is a detriment and the wax occlusion is of small benefit.

GENERAL DISCUSSION OF BONE TRANSMISSION OF SOUND.

That vibration of the whole cochlea is not essential, if indeed it plays any part in the reception of bone transmitted sounds, is indicated in various ways. The affecting of air transmission and of bone transmission in a similar way by the changes of pressure on the drum membrane proves at least part of their paths to the end organ to be identical. It also demonstrates that the ossicles, perhaps also the drum membrane itself, enter into the bone transmission. The filling of the ear with water with resulting increase in efficiency of bone transmission leads to the same conclusion. This is even more strikingly confirmed by the results with wax occlusion, essentially the Weber-Wheatstone phenomenon. These obstructions indicate that the transference of the bone activation to the usual auditory path of air conducted sounds takes place to some extent in the drum membrane, probably through its peripheral attachments, if not in the external auditory canal. For while the pressure effects exerted an influence on the drum membrane and through it on the ossicles, and the water did likewise in addition to filling up the external canal, the occlusion with wax affected not only the external canal and the walls, but also the firmer structures underlying the lining of the walls. That the effects with wax occlusion were not caused by pressures produced on the drum membrane through massaging in the wax is shown by our data on partial occlusion with a hole in the wax. Our data on the effect of pressures also indicates that pressure produces a decrease instead of an increase in bone acuity.

The fact that the bone transmission takes place by means of the outer parts of the air transmission apparatus serves to strongly emphasize the fact of the very perfect acoustical insulation of the inner ear. It is not conceivable that the temporal bone is not set into vibration, especially when the activation is on the mastoid. The insulation effect must therefore be found in the tissue between the bone and the fluid of the inner ear or else in the mechanism of the production of sensation. In the latter case this mechanism would have to be such that vibrations of the fluid would be ineffective in producing the sensation of sound, unless these vibrations were produced only at the oval window.

Some of the theories of the frequency selectivity of the sense of hearing are based on the relative positions of the oval window, the round window, and the different portions of the basilar (or tectorial) membrane to the end organ. A vibration applied to the whole bone might conceivably violate the necessary relationships for a correct frequency to register, but a sensation of sound of some kind should be perceived on the basis of these theories if the whole otic capsule is shaken. It may here be noted that in these bone transmission experiments no change of quality or of frequency was found when the vibration was received by bone conduction as compared with the sound heard by air conduction when the bone receiver was held to the ear. Such a change of frequency or quality might have been expected as indicated above.

It does not seem probable, however, on the basis of any theory of frequency discrimination, that the fluid of the inner ear could be agitated at audible frequencies and not give rise to the sensation of hearing, even though the agitation did not originate at the oval (or round) window. It appears therefore that the insulation effect must be credited to the tissues surrounding the fluid. The efficiency of this protection is shown by the fact that the amplitude of motion of the air particles of a sound wave may be as little as one one-thousandth of one one-millionth of a centimeter (less than one-tenth the diameter of a molecule), and the sound may still be heard. The corresponding minimum necessary amplitude of motion of the drum membrane, ossicles or oval window under these conditions will not be greater than this. Yet the mechanism capable of perceiving these extremely small amplitudes of motion is not ruined or injured when a person receives a blow on the head, and in fact no great sensation of sound is produced by such a concussion.

In the light of the experimentation to date, it seems reasonable to assume that the inner ear is well protected from direct agitation through bone transmitted sounds, and that these sounds reach the inner ear through at least part of the same mechanism of the external and middle ear as is used by air transmitted sounds. This applies to normal hearing people.

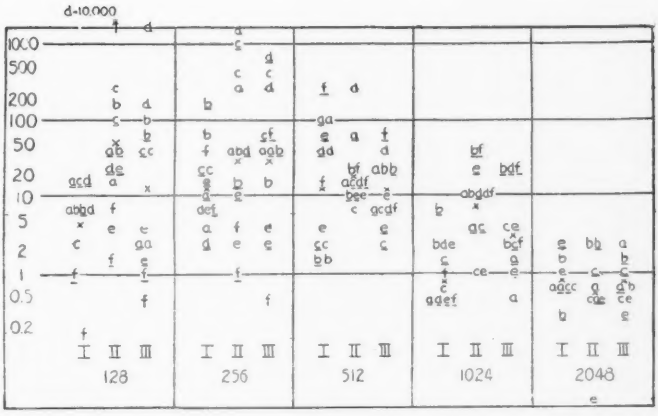

Fig. 1.

Improvement in bone conduction due to water in one ear.

I. Tests from forehead.
II. Tests from mastoid of closed ear.
III. Tests from mastoid of open ear.

Underlined letters refer to the condition of the right ear filled with water.

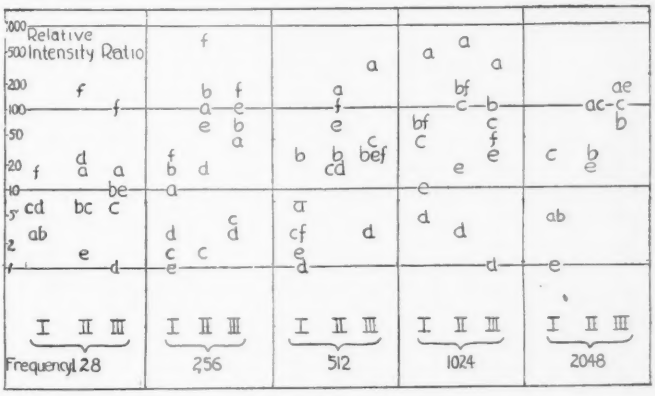

Fig. 2.

Sensitivity to bone conducted sounds.

I. Tests from forehead.
II. Tests from right mastoid.
III. Tests from left mastoid.

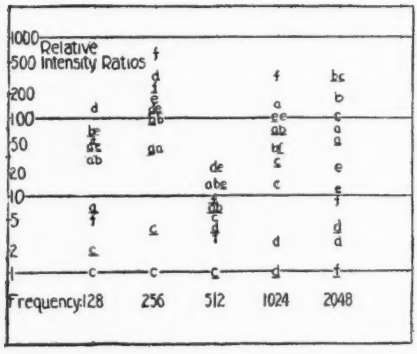

Fig. 3.
Sensitivity to air conducted sounds.
Underlined letters refer to the right ear.

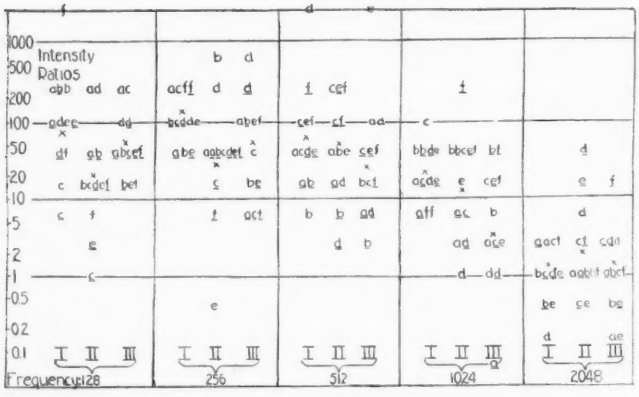

Fig. 4.
Improvement in bone conduction due to wax occlusion of one ear.
I. Tests from forehead.
II. Tests from mastoid of closed ear.
III. Tests from mastoid of open ear.
Underlined letters refer to the condition of the right ear occluded with wax.

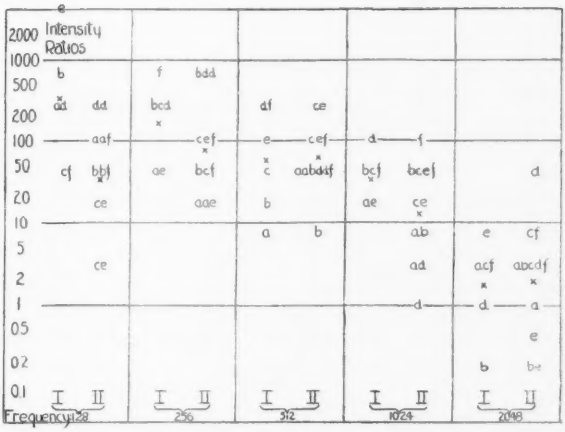

Fig. 5.

Improvement in bone conduction due to occlusion of both ears with wax.

I. Tests from forehead.
II. Tests from mastoids.

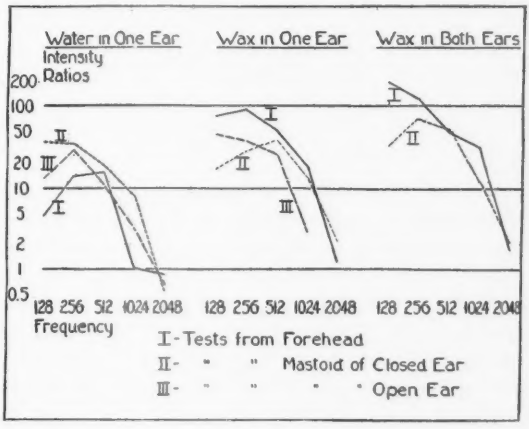

Fig. 6.

Average values of improvement in bone conduction due to closing of ears.

XLI.

ACOUSTIC FILTERING.*

By G. W. Stewart,

Professor of Physics, University of Iowa,

Iowa City.

Occasions will arise when one desires to control sound waves by the equivalent of a process of filtering. This paper is a statement of the present means of filtering sound.

OLD METHODS.

There are at least three well known methods of reducing or altering the passage of a sound wave through a conducting tube, herein called a conduit: (1) One can join a resonator[1] at the side of the conduit and thus eliminate a single frequency; (2) one can use absorbing material in the conduit itself; and (3) constrictions or expansions can be made in the conduit. The action of these three methods will not be here described in detail. None of them nor all of them will comprise a method of altering the transmission of sounds in a conduit at will. For example, it is impossible with them to produce the transmission of a narrow range of frequencies or to produce silence over a broad range of frequencies.

A NEW METHOD.

The existence of an electrical wave filter suggested to the writer the possibility of the construction of an acoustic wave filter. Its technical aspects have been the subject of several articles.[2] An explanation will be given of the action of these filters and a concrete illustration will be presented of the method of making a filter that performs in accordance with specifications. There are three general types of acoustic wave filters shown in the accompanying Fig. 1. H is a high frequency pass filter and consists of a conduit containing equally spaced orifices. It will refuse transmission of all sound waves

*An address given before the Dean Clinical Society at Iowa City, February 8, 1926.

below a certain region of frequencies and will transmit freely all waves above this frequency region. This result is shown in Fig. 2, where the curve represents actual tests upon the filter which is therein drawn to scale.

L is made by enclosing these equally spaced orifices by chambers of equal volume. The result is a low frequency pass filter. An experimental curve of its performance is shown in Fig. 3.

As might be expected, if a combination of these two types is made, the result is a transmission of a single band filter. The performance is shown in Fig. 4.

The action of these filters depends upon the dimensions. Because of the fact that sound waves are not very long in comparison with these dimensions, with the two latter filters there is usually at high frequencies a return of the transmission. This is shown clearly in Fig. 4. This return of the transmission is not a serious obstacle, because it is possible to use several filters in series. In Fig. 5 is shown an example of the production of a single band with practically zero transmission both below and above the desired band. This experimental curve was taken by the use of a single band and a low frequency pass filter in series. The remarkable thing is that the sound transmission above the band is inaudible for many octaves, and this means that the intensity has been reduced probably several million times.[3]

EXPLANATION.

The case of a single orifice in a tube will lead to a practical understanding of the action of these acoustic wave filters. In Fig. 6 is given the experimental values of the transmission of sound by a conduit having a single orifice. At first glance the reader will assume that the reason for the poor transmission at low frequencies is the leakage of sound through the orifice out into the open. But this is not correct. Very little sound actually escapes from the orifice. The motion of the air in the orifice does cause a little radiation from the hole into the open, but very much more importantly it sets up a reflected wave which interferes in the conduit with the oncoming wave. This reflected wave is practically, in fact almost entirely, the cause of the diminution of transmission of sound at the low fre-

quencies. If an orifice will cause such a reflection, a second orifice will also be a source of reflected wave. Thus, if one has a series of orifices he will have a series of reflected waves. The result obtained will depend upon the size of each orifice and their distance apart. Viewed from the physical point of view, the result will be exceedingly complex. There is no doubt of the complexity; it prevents one from seeing just how transmission over a large range of frequencies is eliminated. It is interesting to trace the effect of adding orifices experimentally. In Fig. 7 are shown curves of transmission A, B, C and D; A is with one orifice, B with two orifices, C with three orifices and D with many.[4] The result must be viewed as the effect of interference with sound waves.

A CONCRETE CASE.

Two research workers requested a sound filter that would transmit as shown in Fig. 8. They wished to emphasize the high frequencies relative to the low. They specified that the tube should be a quarter of an inch in diameter.

In responding to this request we proceeded as follows: First, with a knowledge of the frequencies at which they wished 100 per cent transmission, we computed what the size of holes and distance apart would have to be in a high frequency pass filter to give a "cut off" at 5,000. We then proceeded to spoil this ideal filter by using only a few holes. We found that three holes would imitate the curve which they suggested very closely, as is shown in Fig. 9. In this case it chanced that the filter was extraordinarily simple and yet it produced the specified result.

CONCLUSIONS.

The above description illustrates in a somewhat explanatory manner the new tool put in the hands of those who wish to filter sounds. It now seems that it is possible to construct a filtering device approximately meeting almost any requirement proposed.

REFERENCES.

1. A technical statement of the action of such resonators appears in the April number of the Physical Review.

2. (1) "Acoustic Wave Filters," G. W. Stewart. Phys. Rev., Vol. XX, No. 6, December, 1922.

(2) "A Variable Single Band Acoustic Wave Filter," G. W. Stewart. Phys. Rev., pp. 502-505, No. 5, November, 1923.
(3) "Predicted Transmission Curves of Acoustic Wave Filters," H. B. Peacock. Phys. Rev., pp. 525-527, No. 4, April, 1924.
(4) "Acoustic Wave Filters: Attenuation and Phase Factors," G. W. Stewart. Phys. Rev., pp. 520-524, No. 4, April, 1924.
(5) "Acoustic Wave Filters in Series," G. W. Stewart, Jour. of Optical Society of America, Vol. 9, No. 5, November, 1924.
(6) "Acoustic Wave Filters; An Extension of the Theory," G. W. Stewart. Physical Rev., pp. 90-98, No. 1, January, 1925.

3. The technical papers, to which reference has been made, show that it is possible to compute the performance of these filters. Unfortunately the application of the theory is too complicated for reduction to specific directions.

4. Curve D would give 100 per cent at 5,000 vibrations, were it not for the viscosity introduced by a number of small holes. In practice, these holes are not usually small and this experimental effect is not common.

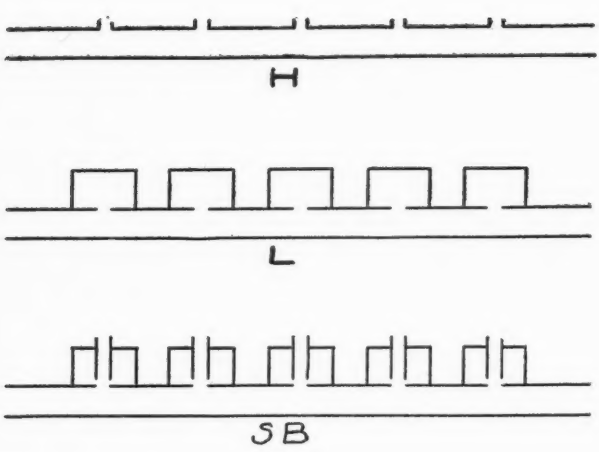
H
L
SB

Fig. 1.

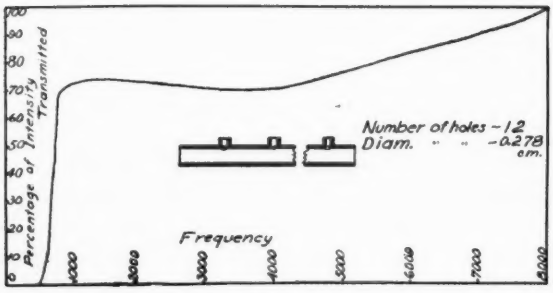
Percentage of Intensity Transmitted
100
90
80
70
60
50
40
30
20
10
0
Number of holes - 12
Diam. " " - 0.278 cm.
Frequency
2000
3000
3000
4000
5000
6000
7000
8000

Fig. 2.

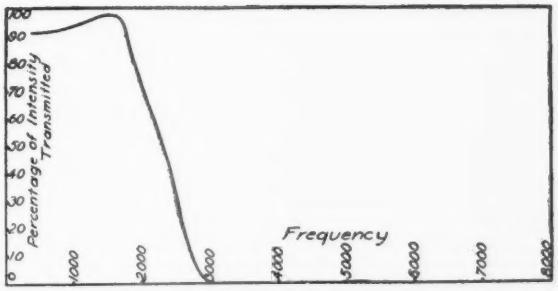
Percentage of Intensity Transmitted
100
90
80
70
60
50
40
30
20
10
0
1000
2000
3000
4000
5000
6000
7000
8000
Frequency

Fig. 3.

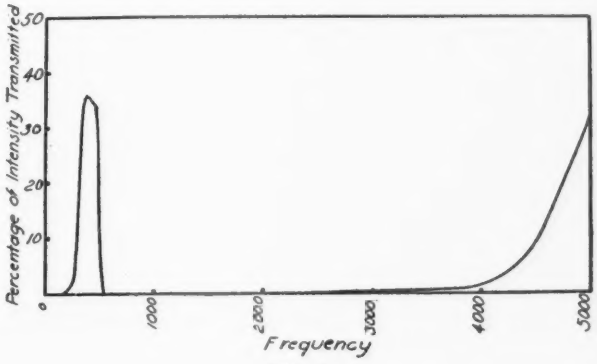
Percentage of Intensity Transmitted
50
40
30
20
10
0
1000
2000
3000
4000
5000
Frequency

Fig. 4.

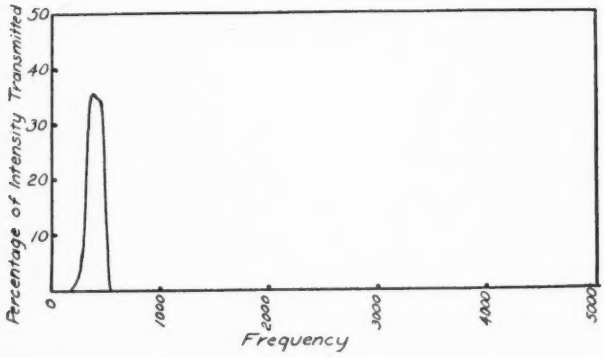
Percentage of Intensity Transmitted
50
40
30
20
10
0
1000
2000
3000
4000
5000
Frequency

Fig. 5.

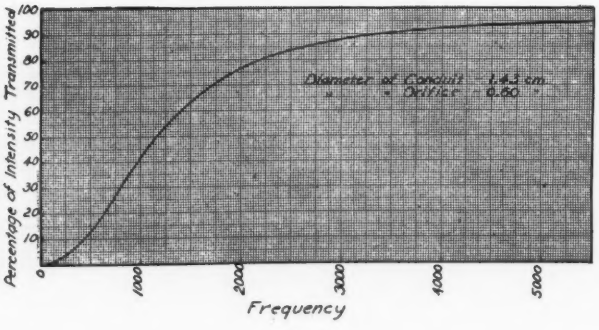
Percentage of Intensity Transmitted
100
90
80
70
60
50
40
30
20
10
0
Diameter of Conduit 1.43 cm
" " Orifice 0.80 "
1000
2000
3000
4000
5000
Frequency

Fig 6.

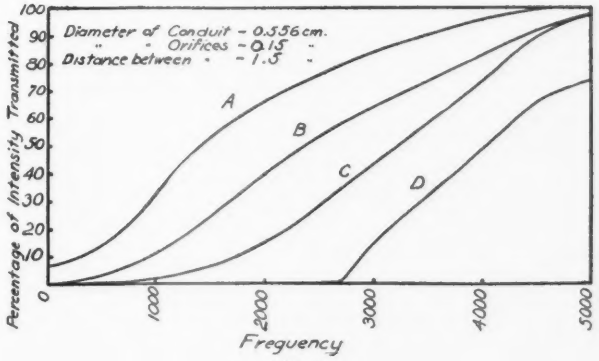
Diameter of Conduit - 0.556 cm.
" " Orifices - 0.15 "
Distance between " - 1.5 "
A
B
C
D
Percentage of Intensity Transmitted
100
90
80
70
60
50
40
30
20
10
0
1000
2000
3000
4000
5000
Frequency

Fig. 7.

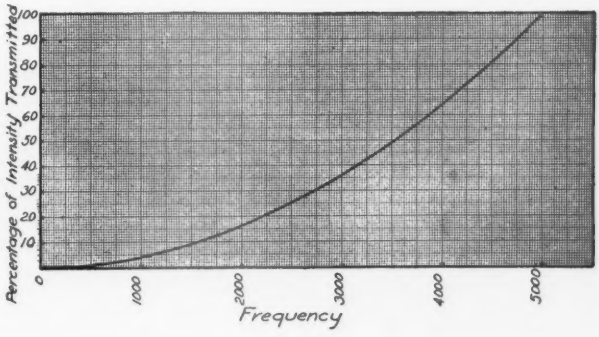
Percentage of Intensity Transmitted
100
90
80
70
60
50
40
30
20
10
0
1000
2000
3000
4000
5000
Frequency

Fig 8.

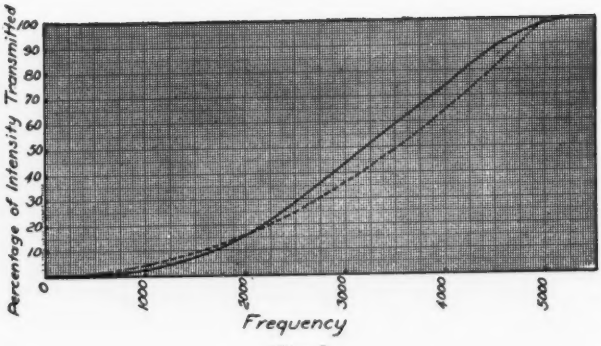
Percentage of Intensity Transmitted
100
90
80
70
60
50
40
30
20
10
0
1000
2000
3000
4000
5000
Frequency

Fig. 9.

XLII.

OTOSCLEROSIS.*

By Dana W. Drury, M. D.,

Boston.

Contributions from the Evans Memorial (no. 127, A-73.)

Part I.

As Gray has aptly said, we are "confronted with one of the most difficult and obscure problems in modern medicine." And because its etiology and essential nature are still largely unknown or in dispute, and its course one prolonged usually over so many years (often with intermissions or natural arrest), to make an estimate of its true value or otherwise of any treatment is a matter of the greatest difficulty. This being so, there is nothing to be wondered at that some otologists hold the view, and frankly tell their patients, that it is an incurable disease.

As knowledge increased, a distinction was made between deafness due to changes in the middle ear and that due to changes in the labyrinth. At this period the disease previously called progressive deafness became known as dry catarrh of the middle ear, owing to the mistaken assumption that the middle ear was the seat of the morbid process. The name was most unfortunate, since the affection is not dry, is not a catarrh, and is not of the middle ear. The mistake was rectified by careful pathologic investigations, which showed that the origin of the symptoms was in the bony capsule of the labyrinth. The disease was then spoken of as otosclerosis, osteoporosis of the labyrinthine capsule, or rarefaction of the labyrinthine capsule. At present it may be defined as an affection characterized by progressive deafness and other symptoms, without the presence of serous or purulent discharge, and, pathologically, by the presence of changes in the bony capsule of the labyrinth, causing in all or most of the cases ankylosis of the stapediovestibular synostosis.

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Valsalva first showed that deafness was sometimes due to fixation of the stapes in the oval window. Morgagni and Meckel also observed the condition. These observations, however, were made in isolated cases, and for many decades no further advance of any importance is to be recorded. Toynbee at length, as the result of the examination of numerous cases, proved that the condition, far from being rare, was extremely common. It was assumed, and quite unjustifiably, that fixation of the stapes was only one of the results of inflammatory action in the middle ear. Moos, in 1861, first published the results of a careful examination of the temporal bone in which fixation of the stapes was found without any pathologic change in the middle ear. In 1885, Bezold associated the clinical features of the disease with the pathologic changes. Katz (1890) was the first to publish the microscopic findings in a case diagnosed during life as a stapes fixation. Politzer (1893, 1901) gave reasons for considering it a primary disease of the bony labyrinth, as the mucous membrane of the middle ear was found to be normal. Specimens were soon noted which showed scattered islets of similar bony changes throughout the petrous bone, with or without stapes involvement. By means of tuning forks and other tests the difficulties of diagnosis have, to a large extent, disappeared, but unfortunately the advance in therapeutics has been very small until a very recent date.

The disease begins by the absorption of bone in some part of the capsule of the labyrinth. The region which is supposed to be most commonly affected is that immediately above and in front of the oval window. Any region of the capsule may be affected, from the edges of the round window to the walls of the semicircular canals. Therefore the symptoms of the affection may vary considerably. The pathologic process consists essentially in absorption of the old bone, followed by the deposition of new formed spongy bone in its place. The diseased focus is at first sharply differentiated from the surrounding healthy bone by a line of demarcation. The Haversian canals within this area become enlarged, their walls being absorbed by osteoclasts. After this has occurred new blood vessels are formed, osteoblasts make their appearance, and new bone is laid down which has a great affinity for staining reagents, being in this respect markedly different from the original bone.

The new formed bone gradually becomes more and more dense, the medullary spaces diminish in number and size, and the new formed blood vessels become smaller in diameter until the bone is again compact. It gradually loses its affinity for staining reagents and, indeed, can no longer be distinguished from the old bone surrounding it.

In the process of absorption the cartilage lining the oval window and the footplate of the stapes, along with the adjacent portions of the annular ligament, disappear and are replaced by bone. Thus it is that the stapes become fixed. There are great variations in the extent to which the stapedio-vestibular synostosis is affected by the process. Sometimes there is merely a thin lamella of bone joining the stapes to the margin of the oval window, while in other cases the walls of the oval window, the annular ligament and the stapes are united into one bony mass. Small exostosis are occasionally found in the margin of the round window, and the oval window is sometimes affected in the same way. It is important to observe that during the whole process there is no sign of inflammatory action.

Local and constitutional conditions may play an important part in determining the onset and course of this disease. Nevertheless, the evidence of heredity plays a very distinct rôle. It is an old matter of experience that progressive deafness occurs in several successive generations of a family. First to draw attention to this fact was von Tröltsch. He believed that in deaf families peculiarities in the structure of the skull were inherited, which favored the onset of the socalled chronic simple ear catarrh. Otosclerosis, which occurs principally in the bone surrounding the labyrinth, invading the tympanum only near the labyrinth wall and its windows, cannot be the result of unfavorable conditions in the pharynx, in the tubes or in the tympanum, and it is the only condition which leads to inherited progressive deafness. Politzer is inclined to regard the hereditary tendency of otosclerosis as of importance in differential diagnosis against the adhesive processes. Biology, however, has developed a theory of heredity which is capable of explaining the much discussed nature of otosclerosis.

The children inherit, or the parents transmit, only those peculiarities which are contained as rudiments in the germ

plasm of the parental sexual cells. The union of the ovum and the sperm cell completes the act of inheritance. The inherited materials of the parental germ cells are called determinants. The usual number of determinants do not alone originate in the plasma of the parents but from the collected ancestral plasma. They may be transmitted from one individual to the progeny without ever having become active in that individual or in part without ever having become later active. What is not contained in the determinants no external influence can add to the individual germ plasm, which is complete after the germ union.

As otosclerosis is apparently inherited, it would seem that it is not a disease, at least in a scientific sense. Otosclerosis would be a disease, and a congenital one, but not a hereditary disease if it were of syphilitic origin, as Habermann assumes. He claimed that it occurred generally between the ages of twenty and forty, a period in which syphilis is most frequently acquired. I doubt very much the correctness of this age. It is true that most of those suffering from otosclerosis seek aid in those years, but E. Hartmann has shown that the onset of this extremely insidious trouble probably occurred at a much earlier date. In reality an early onset of otosclerosis is not rare, and early examples of the disease have been mentioned by Bezold and Scheibe. If, therefore, otosclerosis is not a disease in the true scientific meaning of the word, how are we to regard it? The answer is furnished by the interesting pathologic investigations of Siebenmann.

We call otosclerosis that form of progressive deafness which pathologically rests upon a hyperostotic new formation of bone in the labyrinth capsule. Though many authors believe that these changes are derived from an ossifying periostitis following an infection of the mucous membrane in the tympanum, in reality they are in the oldest parts of the process to be found at the periphery of the primary labyrinth capsule and the connective tissue bone secondarily developed from the periosteum. Siebenmann regards the rarefaction in the last phase as a development process, which normally does not occur in the petrous bone, but is the rule in other bones, and consists in a complete disturbance of the cartilage situated in the interglobular space and in the window margins, and the transfor-

mation of the compact bone into osteoid tissue and finally into spongiosa. This proves the fact of its hereditary nature. Every stage of growth, whether normal or abnormal, must be furnished in a determinant transmitted from the ancestral plasma of the individual, and is therefore heritable. Whether the determinants become active may in our cases depend upon certain influences of an internal or external nature. Clinically, we have seen that the onset of puberty, which is probably the greatest incentive to bony growth, and the child bed, which also causes changes in the bony system, may be regarded as exciting causes.

The normal determinants which preserve the species are constantly transmitted. If this did not take place, man would have degenerated long ago. The determinants which represent individual characteristics are different. These are inherited, but do not become active in each successive generation, but may skip a child, and then again appear in a grandchild. This is spoken of as a latent inheritance. It would, of course, be difficult in these cases to discover the determinants.

The law of inheritance explains the character of otosclerosis as an inherited abnormal process of development. When and how it occurred in our ancestral plasma is, of course, unknown. The presence of heredity, as Hammerschlag has correctly said, simply pushes the question back to the original cause, which is unfortunately at an infinite distance. Though the light which the theory of inheritance throws upon the nature of otosclerosis is slight, we nevertheless receive more information as regards the prophylaxis and treatment of progressive deafness. When the determinant of the abnormal development has become active, every hope of successively combating this affection must be regarded as futile. The prophylaxis is somewhat more encouraging.

It is generally admitted that in otosclerosis the female is more often affected than the male. This is probably due to parturition, disorders of menstruation or prolonged lactation. Further, women suffer more often from anemia. According to Körner, women transmit the tendency more frequently than men, besides being themselves more liable to suffer from it. If inheritance plays such an important part as I believe that it does, it would naturally be expected that race would also

have some effect upon the incidence of the affection. Bezold's statistics show that from 57 to 66 per cent of cases of otosclerosis occur in women, while only 42 per cent of all ear diseases are found in the female sex. Seven per cent of all ear cases suffer from otosclerosis.

According to Hammerschlag, infectious diseases are causative of secondary periostitis and osteitis of the labyrinth capsule. The sclerosis produced by certain infections are limited to vascular areas—e. g., sclerosis due to variola affects the pulmonary arteries. Hence the possibility exists that certain infections may influence the petrous bone in a similar way. Many authors have noted a connection between rheumatism, gout, arthritis and otosclerosis. Bryant holds that though these diseases are often found in cases of otosclerosis, it is more probable that all the conditions are due to a common cause than that they give rise to otosclerosis.

Gruenert, Manasse, Heymann, Heine, Katz, Heimann, Gradenigo and Paludetti all regard syphilis as a predisposing cause. Habermann, who reports a case with marked luetic changes in the vessels, holds that the syphilitic cause of otosclerosis is shown chiefly by the histologic findings of chronic inflammation extending to the bone from the periosteum by way of the vessels, without suppuration or necrosis. The presence of hyperostoses and also the porous composition of the bone favor a syphilitic origin. On the other hand, Bezold, from an experience of over a thousand cases, does not believe in the causal relationship of syphilis. Beck and others have tested the Wassermann reaction in many cases of otosclerosis and found it positive in a very small percentage (five times out of forty-four cases). Gray notes that syphilis is more common in men, while otosclerosis is more common in women.

Scheibe, Heimann, Katz and Habermann consider that the bony lesion in otosclerosis is an inflammatory osteitis. Mayer holds that otosclerosis is closely allied to the osteitis fibrosa of von Recklinghausen. Manasse compares otosclerosis to the vascular osteitis of Volkmann. Bryant says that both in otosclerosis and in osteitis we find increased vascularization, displacement of the normal bone by osteoid tissue, exostosis and hyperostosis, the line of demarcation, or Pommer's cementing line, the origin in the endosteum, or from the periosteum,

and finally resolution by new atypical dense bone. Again, Politzer, Bezold, Siebenmann, Möller, Bruehl and Schwartze hold the view that otosclerosis is an idiopathic primary bone disease. Politzer and Bruehl point out that the otosclerotic foci are separated from the periosteum by layers of normal bone, and that they may even occur in the region of the internal meatus where there is no mucosa. Ankylosis of the stapes is by no means always present. Under normal conditions growth should cease in the labyrinth capsule at or soon after birth, though it continues in other bones. Siebenmann views otosclerosis as the continuance of a process of growth in the labyrinth capsule, in the regions where remnants of embryonic cartilage most abound. Taniyama has found marked remains of the primitive cartilaginous capsule of the otic vesicle in the bone in the anterior margin of the oval window in the fetus at various ages, and also in children and even in adults.

As to vascular theories, O. Mayer, Cornet and Gray have all wholly or largely given up their earlier theories as to the origin in local anemia, although Gray believes that the foci of disease in the temporal bone represent a process in which minute portions of bone have died from insufficient nourishment and are replaced by new bone. In support of this opinion is the existence of a sharp line of demarcation round the affected part and the absence of all inflammation. In this connection Bruehl states that the diseased areas present the appearance of an enucleable foreign body. Cornet reports that 50 per cent of his otosclerotics had arteriosclerosis. He mentions the fact that on microscopic examination there is atrophy in the organ of Corti, just as in senile deafness due to arteriosclerosis. In this connection it has been noted, in cases of senile deafness which are due to changes in the labyrinth or auditory nerve, that it makes its appearance earlier in those who are already the subjects of otosclerosis. O. Mayer has found atheroma in the internal carotid and auditory arteries in otosclerosis. This cannot account for the disease in young people who have no vessel degeneration.

Ferreri says that in many cases of congenital deafmutism the loss of hearing is due to an early attack on the capsule in fetal life. This attack is coincident with maternal intoxication or other affections, such as rickets or osteomalacia. All the osteo-

malacic patients seen in Arcangeli's clinic had evident otosclerosis. The administration of a vaccine improved the osteomalacia and also the otosclerosis. Ferreri claims also that rachitic lesions of the labyrinth capsule can be recognized in the newborn. I believe that rickets and osteomalacia have been proven etiologically the same. Alexander has found otosclerosis in cretins (human and animal).

Severe psychic traumatism—e. g., shock, fright, grief, anxiety, mental exhaustion and neurasthenia—is held by some authors as the cause of otosclerosis. Baginsky believes that a nerve lesion is the primary cause. Manasse states that in almost all cases nerve changes have been found in the inner ear on microscopic examination, even in cases where the foci of diseased bone were very small. Others hold that the nerve changes in the labyrinth are due to the bone lesion (Siebenmann). Froeschels states that the diminution or absence of the sensibility to tickling in the external auditory meatus indicates the presence of a nerve lesion. According to Panse, four cases diagnosed as otosclerosis were found on examination to have degeneration of Corti's organ and rigidity of the stapes but no bone lesion. J. Möller states that constant exposure to noise or traumatism may be the predisposing or exciting cause of otosclerosis. O. Mayer points out that vasomotor neuroses may follow trauma. According to Bruehl, the anterior margin of the oval window is especially vulnerable on account of the greater movement of the anterior part of the footplate of the stapes, and also because of the presence in this region of the pulley of the tensor tympani muscle. Gray adds that in addition the stresses set up by the continual stapes vibrations sensitize the bone in the neighborhood to new formations or pathologic changes.

According to Katz, the scrofulous constitution bears a causative relation to otosclerosis. Several other authors have felt that it was directly influenced by tuberculosis. Again, Politzer was the first to suggest the relation of otosclerosis to the puerperum. He thought it was a true infective puerperal osteitis. In this connection, Mayer believes that there is some connection between otosclerosis and pregnancy, as during the time there are other bony outgrowths, the socalled puerperal osteophytes. Stein believes that in pregnancy, otosclerosis may

begin or be aggravated. He thinks that the first pregnancy is the most vulnerable.

Therefore, I offer as a suggestive etiologic factor in otosclerosis the assumption that it (otosclerosis) begins about the time of puberty. We know that youth is the plastic age. During this period the cells that are on their way toward specialization may, if conditions become altered, especially in a predisposed person, dedifferentiate, becoming thereby embryonal again; and then they start off on a new line of differentiation. For example, an individual with the correct determinants, through a chemical change in the body, possibly controlled in activity by the endocrin glands, passes towards puberty, then the embryonic cells again appear, influenced possibly by the persisting embryonic cells remaining in the cartilage, differentiates into a new bone formation in the labyrinthine capsule. This later process we will call regeneration. Botanically, if, in mowing, a young ragweed is decapitated, its parenchymal cells dedifferentiate into meristematic cells again, and these set about forming a new top by redifferentiation into all the types of cells normally occurring therein. Zoologically, the egg of a frog normally divides into two cells, each of which respectively gives rise to different tissues of the adult amphibian. But if we shake apart these two blastomeres each will give rise to a distinct frog: for the cells are still in their period of juvenescence and have the ability to return to their original state and start development over again along new lines. Now, if the same individual had been a little older, dedifferentiation, and hence regeneration, would have been impossible, in spite of the determinants, for maturity would already have set in.

The ultimate degree of the deafness may be either comparatively slight or very great, but only in rare cases does it become so marked that the patient cannot understand a loud conversational voice in close proximity to the ear. Pain is conspicuous by its absence, and even when present has probably no relation to the disease. Second to the deafness are the subjective sounds. In some, tinnitus is more distressing than the deafness. It has been tried to classify the tinnitus but without success. In some it is intermittent, in others it is continuous, and in all the intensity varies in degree. Want of mental

occupation makes it more noticeable, and if the attention is taken, tinnitus is less marked. The surrounding silence of night makes it more noticeable, as does the use of alcohol and tobacco. Those of an optimistic turn complain but little, while the introspective may try suicide. The cause of the tinnitus is probably the change in the physical conditions to which the intralabyrinthine fluids are subjected. Fixation of the stapes must, to a certain extent, interfere with the compensation required to allow for the changes of tension to which these fluids are subjected during the cardiac systole and diastole. Hence the changes of pressure during the phases of cardiac activity will be brought to bear upon the organ of Corti more than in normal circumstances, and the consequent stimulation of that structure will produce the subjective sensation of sound.

In many persons who have been deaf for a long while, a change in the physiognomy is frequently to be noted. For several years after the onset of the deafness a strained look is usually, though not always, present, and this is apt to be accentuated if tinnitus is a marked feature. As time passes, and particularly if the deafness increases and the tinnitus becomes less irritating, the strained look gradually passes into one of calm serenity.

The diagnosis of otosclerosis in its typical form, when associated with progressive deafness, can be established with certainty. If, in a case of progressive deafness, the membrana tympani is normal, with or without a reddish luster from the inner tympanic wall, the eustachian tube permeable during catheterization, Rinne's test negative, the low tones inaudible and the perception limit for the highest tones present, it is evident that the case is one of otosclerosis. In the initial stage of the affection, where the deafness is slight, and where the subjective noises are the prevailing symptoms, we cannot say with certainty whether we are dealing with otosclerosis or with nerve deafness. In such cases, therefore, if we find a hereditary predisposition, we may suspect otosclerosis.

The diagnosis is made with more difficulty in those numerous cases in which the membrana is cloudy, atrophic or retracted, and where catheterization shows that the tube is swollen and narrowed to a varying degree. Under such circumstances, the differential diagnosis between otosclerosis and

catarrhal adhesive processes of the middle ear often remains undecided. This is easily understood, if we consider that the latter, when combined with calcification and ossification of the annular ligament of the stapes, adhesions of its crura with the walls of the recess of the oval window, and with thickening of the membrana fenestræ cochleæ, can run their course with symptoms of progressive deafness, and that the functional tests may give the characteristic results of a marked hindrance to the sound conducting apparatus, just as in otosclerosis.

We may assume, on the other hand, with great certainty that otosclerosis is the anatomic cause of the deafness if we find the membrana tympani only cloudy, the eustachian tube of normal permeability, the disturbance of hearing progressive and the results of the functional tests characteristic. If the membrana is retracted, especially in conjunction with impermeability of the tube, the diagnosis is uncertain. In such cases, we may assume that the catarrhal adhesive process in the middle ear is complicated with the affection of the labyrinth, only if the disturbance of hearing is of a marked degree, and is accompanied by continuous subjective noises, and a shortened perception for the tone of the tuning fork through the cranial bones.

If the stapes be fixed there will be no difference in the intensity of the sound during compression and relaxation of the air in the external canal wall (Gelle), whereas if the stapes be free the sound will be diminished during compression as in a normal ear. Now, with Schwabach's test, the duration of bone conduction in the patient is compared with that in a person with normal hearing. If the bone conduction is longer in the patient, then he is presumed to be suffering from middle ear disease. As to Bezold's triad, it is unfortunate, however, that in many cases of undoubted otosclerosis the triad of symptoms is incomplete. Rinne's test is not always negative, and the bone conduction is not always prolonged. This is the result of changes in the labyrinth or in the auditory nerve in its bony channels. Paracusis Willisii is frequently observed as is vertigo, but it is observed in other diseases of the ear as well. The reason of the improvement in a noise is a shaking up of the ossicles, whose joints have been rigid; these bones are thus thrown out of their equilibrium, and are thereby

rendered more capable of transmitting the waves of sound. It has already been shown that the terminations of the acoustic nerve are also simultaneously thrown out of their equilibrium, which causes them to become more easily excited by the waves of sound.

The treatment for this disease has been varied and of little value to the individual. Many things have been tried, each with some degree of acclaim for the time, but soon being discarded as valueless. Local treatment through the eustachian tube is probably harmful, while massage by the way of the external auditory canal gives more relief, this applying when the condition is just beginning and there is some mobility of the ossicles, especially the footplate of the stapes. The operative procedures which were formerly practiced to a great extent did not fulfill the expectations attributed to them, and the favorable results which at first were ascribed to them have dwindled to a minimum. These operations are: (a) mobilization of the stapes, (b) synechotomy of the cura of the stapes, (c) excision of the entire membrana tympani with the malleus and incus, (d) finally extraction of the stapes. It is suggestive that in the early stages radiation might be of value, as we know its good effects on growing cartilage and bone in young animals. Webster and Siebenmann are both at work on this line of treatment. Joseph, on the other hand, found that diathermy was harmful to an otosclerotic.

PART II.

It is generally accepted that the internal secretions of the ductless glands are in some obscure way correlated with the general condition of the organism. For present purposes, it may be sufficient to postulate that in certain measure the general metabolism of the organism is regulated by these secretions. The endocrine glands influence the harmonic development of the body; they are among the directing factors of morphogenesis. The different parts of the body do not develop in an autonomic way wholly independent of each other, but during growth they stand in continuous if quantitatively varying interrelation. By virtue of their functions the endocrin glands are controlling factors of development, each morphologic feature being subject to their influence. It is clear, there-

fore, that these organs are of the utmost significance for the ontogenesis of man. While the whole endocrine system is not an organic unit, in its several elements it exercises a large influence over our somatic organism. The endocrine system might be described as the sense organs of the autonomic system, sensitized primarily to chemical stimuli as are the sense organs of the cerebromedullar system to those of physical origin. These organs judge the quality and intensity of action of the chemical substances produced in the body and regulate the nature and quantity of the same by means of their harmonic activity. Our steadily growing knowledge of the effects produced by these internal secretions has given to the medical profession valuable methods of treatment, but even today the danger exists that the treatment will outstrip our knowledge, and enthusiasts pin their faith much too largely—yes, even injuriously—on an indiscriminate pluriglandular therapy.

For example, it has been demonstrated that primarily the pancreas, and in lesser but significant degree the pituitary, the thyroid, the suprarenals and the ovaries influence carbohydrate metabolism. The thyroid seemingly has an important effect on protein metabolism which is not shared equally by the other members of the group. An increased thyroid function connotes a higher level of protein metabolism, while a lowered function correlates in a diminution of the nitrogen exchange. The parathyroids apparently are intimately concerned with the calcium metabolism, but the exact nature of the mechanism is still unknown. The gas exchange of the organism is profoundly influenced by the functional level of the ductless glands, most strikingly shown in the disturbed functions of the thyroid. These and many similar facts, established and proven by experiment, have formed the basis of certain objective methods for the determination of the functional capacities of the several glands composing the endocrine group (Rowe). Were it not for the fact that the performance of the necessary tests is so time consuming, complex in detail and requires so extensive an instrumental equipment, the examination of these processes as aids to the diagnosis of the endocrinopathies would have a much wider application.

The thyroid gland is indubitably of great importance in the human, and marked aberrations of the functions of this

gland give rise to symptoms which may be definitely correlated with well recognized syndromes. In like manner, the adrenals and the two lobes of the pituitary determine clinical entities which in their later stages are readily recognized. Studies of male and female castrates have determined also certain fairly well defined pictures which equate with those obtained where the glands in vitam have undergone pronounced functional involution. Growth anomalies are produced severally by the thyroid, the anterior lobe of the pituitary and presumably the gonads. Certain forms of obesity are also referable to disturbed function of the several members of the group. Instances could be multiplied indefinitely, but the above offers a sufficient background for the subsequent discussion.

THE THYROID.

In 1902, Frederick experimented with thyroid extracts and reported negative results except in cases where myxedema and deafness were coincidentally present. Tweedie, in 1908, assumed that otosclerosis was attributable to some influence of the thyroid, either by direct effect on the nervous system or through alteration of the normal condition of the blood and circulatory mechanism. In this connection, Bryant, in 1911, believed that an etiologic factor of otosclerosis was a trophic and toxic disturbance due to an altered internal secretion of the thyroid. Vulpius stated that the lessened aural acuity of many cases of myxedema was essentially improved by the routine administration of thyroid preparations. Subsequently he introduced thyroid treatment in handling cases of adhesions in the middle ear. Bruehl studied twenty-one cases of otosclerosis in Politzer's clinic. From his results he maintained that the trial of thyroid extract was indicated in every middle ear process where the changes existing were of a reversible character. In cases of stapes fixation, he regarded the treatment as devoid of any prospect of betterment. A similar conclusion was reached by Alt and Eitelberg in Grueber's clinic.

In certain cases of goiter Manasse observed bony changes in other portions of the body similar to those reported in otosclerosis. Hoennicke in this connection observed that epidemic osteomalacia always appeared in a goiter district and that of the many cases examined by him only one gave no tangible

evidence of thyroid disease. On the other hand, Senator takes exception to these findings and asserts that a thyroid etiology is not implicit in all otosclerotics, as more extended metabolic studies indicate that other endocrine glands, particularly the ovaries, may exercise the same effect as the thyroid. Axhausen holds that changes in the internal activity of the ductless glands, but primarily in the thyroid, determine the processes of bone dystrophy. He confirms Hoennicke in an intrinsic connection. McCrudden's interesting studies on the etiology of osteomalacia might well be adduced as support of the views of Senator. Manasse, as well as Siebenmann, Alexander and Mayer, have observed improvement in cases of otosclerosis under thyroid medication and report similar results in the cases of deafness associated with cretinism. Peltesohn, working with Hammarschlag in Politzer's clinic, ascribes the beneficial effect of thyroid medication to the iodin content.

In 1921 Frey noticed changes in the thyroid, especially in otosclerotic females, during their normal sexual life, such as the pubescent period and that of pregnancy. Ratner's earlier investigations were concerned with the loss of hearing in cases of thyroid dysfunction and the favorable therapeutic effect of thyreodin on the aural acuity. Stimulated by his results, he sought to establish a definite connection between otosclerosis and thyroid dysfunction. In 60 per cent of all his cases of deafness he believed he observed changes in thyroid function which were usually hyper- and dys- activities in women and hypothyroidism in men.

THE PARATHYROIDS.

Bloehmke suggests the possibility of connection between the bony changes found in otosclerosis and the functional activity of the parathyroids. He suggests that attention should be given in cases of otosclerosis to the postmortem examination of these glands. From the clinical standpoint, this view was elaborated by Frey and Orzechowski. In the course of a study on latent tetany, primarily in relation to certain other pathologic conditions, they considered the question of a direct equation with otosclerosis. Frey examined cases diagnosed as otosclerosis who also exhibited tetany, but failed to draw any conclusions. Leicher states that in most cases of otosclerosis

there is a small but established diminution in the serum calcium. To raise the total calcium content he recommends the exhibition of phosphorus for months or years coupled with homeo-or hetero-plastic transplantation of the parathyroid. Siebenmann's treatment with phosphorus was based on his belief in an etiologic connection between otosclerosis and osteomalacia. Kobrak supported the views of Leicher in regard to calcium, while Cornet felt that the causative element was a chlorid retention rather than a calcium loss.

THE PITUITARY.

Citelli held that otosclerosis might be caused by the infections of the endocrine glands in general and of the pituitary system in particular. He felt that the administration of pituitary extract produced good results. Eckert, Pollock, Pratt and Peltesohn all observed improvement with pituitary extract. Pollock and Pratt first studied each case for the existence of a focal point of infection, and if none were determined then exhibited pituitary. Lermoyez believes in the possibility of pituitary involvement in cases of otosclerosis; he regards the use of thyreodin as potentially harmful; he concludes that pituitary extract is never harmful and, by causing a phosphate retention, may be of indirect advantage to the case. Fraser, on the other hand, states that he has never observed improvement in the use of gland extracts in cases of otosclerosis and compares their exhibition to "the match that lights the fire," hereditary tendencies and the female sex corresponding to the "inflammable material."

Kobrak, Caldera and J. C. Beck believe a relationship to be established between otosclerosis and hypofunction of the pituitary. Beck also includes the suprarenals as complementary factors. He reports successful treatment in seven cases in a group of eleven. Bossi refers osteomalacia to disturbances of the adrenal and reports cases occurring in pregnant women cured by adrenal exhibition. He emphasizes the influence of the adrenal in the processes of ossification. Stumme and Erdheim, Lancaster and others have observed an increase in the pituitary body during pregnancy and a return to the normal after confinement. From this standpoint, Denker traces these hypophyseal changes to a lowered function of the ovaries, and

states that gonad extracts may hinder pituitary hyperplasia. He suggests a possible connection between tumors of the hypophysis with or without acromegaly and otosclerosis. Lately he has endeavored to establish in the blood serum of otosclerotics the presence of a protective ferment against pituitary products. He questions if all cases of otosclerosis should be referred to the ductless glands. Moraczewski, working in Denker's clinic, has demonstrated retention of phosphoric acid in otosclerotics by the exhibition of hypophysin tablets.

THE OVARIES.

Many authors believe that the gonads play an important part in the etiology of otosclerosis. Overactivity of the ovaries is given as the cause of osteomalacia and the curative influence of ovarectomy emphasized. This point of view ignores the most important and informative studies which have been conducted by McCrudden on the etiology of the latter condition. Stein, with a series of 125 cases of otosclerosis, two-thirds of whom were women, selects forty cases to illustrate the thesis that the otosclerotic processes proceed most rapidly in women with menstrual irregularities. He regards the presence of genital anomalies as implying unfavorable prognosis. Two of his cases showing particularly rapid progress were, respectively, a case of infantilism and an asthenic girl with cardiovascular disturbance and a hyperplastic uterus. Of ten male patients developing otosclerosis at puberty, two had a morphologic hypoplasia, one a hyperplasia of the genitals and one case of hypospadias. He cites cases in which sexual excitement increases subjective noises to the point of interference with normal sex life. He lists cases of neurasthenia sexualis showing a wide variety of manifestations. It is perhaps somewhat unwarranted to ascribe many of the conditions thus cited to internal secretory disturbance of the gonads.

Frey cites a case where aggravation was produced by the impotence of the husband and another by excitement. Both Stein and Frey believe there is a distinct connection between the gonads and the ear through a nutritional control and, with disturbances of the former, the normal building processes are disturbed and the vegetative system affected.

In a study of the connection of otosclerosis with disturbed

function of the endocrine system covering a period of two years and involving careful observation and study of a series of over a hundred cases, the writer has found that fully one-third of all of the series gives neither subjective nor objective evidence of any endocrine disturbance. Of the residual two-thirds of the cases cited, endocrine foci have been more or less definitely incriminated and severally include the thyroid, the pituitary and the gonads. The absence of adrenal cases from the group may as well be ascribed to the paucity of cases presenting disease of these organs as to a failure of the influence analogous to that established for the other ductless glands. The conclusions drawn from these studies and from other series of observations by the writer connected with investigations of the entire topic would be that the endocrine influence is an indirect and nonspecific one. As was before stated, the endocrine system in its several components plays a most important part in the general regulation of body metabolism. Functional failure of any endocrine element as well as disturbances of a nonendocrine origin may upset the myriad of delicately adjusted equilibria and produce otosclerosis as an end result of the same. That there must be a constitutional element, a diathesis, a tendency, is intrinsic in the fact which has been demonstrated that neither do all endocrine cases exhibit otosclerosis nor are all otosclerotics endocrine cases. It seems fair to state in this connection that as other tests are developed and proven of worth, chemistry is destined to tell us much of importance as causative factors in otosclerosis.

Space permits the presentation of only two cases from the present series which illustrate certain of the points touched on in the foregoing discussion. The patients are boys, brothers, the older sixteen years of age, and the younger fourteen.

Family history: The father is living and is described as of a nervous temperament. The mother is also living and well, although of a definitely obese physical habit. The two children in question have four living siblings, some of whom present otosclerosis. Two other siblings died shortly after birth and two at premature birth. The family history is free from record of arthritis, diabetes or cancer. On the paternal side there is an uncle who exhibits the same type of deafness and is now under treatment for the condition; while on the mother's side

both her mother and her maternal grandfather exhibited the same type of deafness.

Certain details of the two cases must be discussed individually.

Case B-379.—This is the younger boy, 14 years of age. His medical history is negative except for an attack of measles when very young. Some two years ago his deafness became manifest and has progressed rapidly to the point of serious impairment. His disability has caused him to become very quiet and retiring. His habits are excellent, his disposition good. He is apparently keenly alive to the situation and is studying lip reading assiduously.

Case B-380.—This is the older lad, 16 years of age. His previous history shows an attack of measles at a very early age, an operation for right inguinal hernia at the age of 7, and three tonsil operations. At the age of 13 he received a serious injury to the right hip through an accident while coasting. Both ears have discharged intermittently in the past. He has now a negative Rinne in both ears, the stapes are not fixed, and there is prolonged bone conduction and marked paracusis Willisii. Both drum membranes are healed. Physical examination shows a huskiness of voice; mouth, tongue and throat negative; the neck is large but without any glandular hyperplasia; there is a marked tendency to obesity. In character he portrays a definite extreme from the other brother. There is a dull mentality and an incorporative and unhelpful attitude. He sleeps and eats well, but is sluggish mentally and physically. From the following tables the following facts are noted:

TABLE I.

PHYSICAL MEASUREMENTS.

	B-379	B-380
Age	14	16
Height	149	164.5
Sitting height	81.5	88.
Sitting height index	0.540	0.535
Chest	72	93
Weight	40.1	69.8
Area	1.29	1.76
Lung capacity	2600	3600

The several physical measurements are given in Table I. They illustrate primarily the disparity in size between the two boys. The sitting height indices are both a little high, but not outside the normal range of variation. The older boy's lung capacity, although absolutely much larger than the younger's, is relatively not increased.

TABLE II.

VITAL CAPACITY.

	B-379	B-380
Weight	+15 per cent	+17 per cent
Chest	+ 6 per cent	+11 per cent
Vital capacity	— 6 per cent	—13 per cent

Table II contains the data secured by comparison of observed results with the values predicted by the Dreyer formula. The experience of this laboratory has shown that only the standards referred to sitting height are to be regarded as significant. The Dreyer concept postulates a relatively normal body configuration and in cases of obesity or marked emaciation leads to extrapolations which are most misleading. The younger boy, as will be seen here, is underweight. The older is somewhat obese. The vital capacities fall within the normal range, although that of the older boy is possibly of a borderline value.

TABLE III.

URINE FINDINGS.

	B-379	B-380
Volume	860 cc.	2200 cc.
Specific gravity	1.015	1.012
Albumin	0	0
Casts	0	0
Sugar	0	0
Urobilinogen	0	0
Salol	75′	75′
P-S-P, first hour	34%	40%
second hour	12%	13%
Total	46%	53%

Table III contains the significant urine findings. The freedom of both cases from abnormalities is potentially significant.

They both show a somewhat low phthalein value. The salient difference is in the volume of the urine.

TABLE IV.

NITROGEN PARTITION.

	B-379	B-380
Total nitrogen	6.85 gms.	13.43 gms.
Urea nitrogen	82.5%	79.6%
Uric acid nitrogen	1.09%	2.0%
Ammonia nitrogen	3.1%	3.2%
Creatinin nitrogen	3.6%	4.7%
Residual nitrogen	8.9%	10.5%

The nitrogen partition values show an apparent disparity in the relative protein level. In view of the fact, however, that the older boy is almost double the weight of the younger, the disparity is more apparent than real. The residual nitrogen of the younger boy shows an upward tendency, of the older boy a value well above the conventional normal.

TABLE V.

GALACTOSE TOLERANCE.

	B-379	B-380
Positive	20 gms.	10 gms.
Negative	10 gms.	5 gms.

The differences in the sugar tolerance, as shown in Table V, are striking. The younger boy shows a depressed tolerance, it is true, but one of relatively small magnitude. The older boy, on the other hand, exhibits a significant diminution in the tolerance level. These two tests incidentally illustrate the misleading character of tolerance studies where dosages are based on body weight.

TABLE VI.

BLOOD MORPHOLOGY.

	B-379	B-380
Hemoglobin	100	95
Erythrocytes 10^6	7.22	5.90
Leucocytes 10^3	11.6	8.4
Polymorph. neutrophiles, %	61	56
Lymphocytes	35	36
Eosinophiles	2	6
Monocytes	2	2

Sharply divergent are the morphologic findings of the two bloods. The younger boy shows a polycythemia, a slight leucocytosis with an approximately normal differential partition. The older boy, on the other hand, shows a normal red and white count with an established eosinophilia.

TABLE VII.

BLOOD CHEMISTRY.

	B-379	B-380
Nonprotein nitrogen	38	32
Urea nitrogen	16	13
Uric acid	3.9	4.5
Creatinin	1.5	1.6
Residual nitrogen	20.1	16.9
Sugar	91	99
Wassermann	0	0

In Table VII the salient differences are the higher nitrogen values for the younger boy and the relative normalcy of the older, with the single striking exception of his blood uric acid. There is a suggestion in the blood chemistry of the younger lad of a possible incipient kidney condition. That of the older patient shows no such tendency.

TABLE VIII.

RESPIRATORY METABOLISM.

	B-379	B-380
Basal metabolism, rate	1410 cal.	1521 cal.
Per cent	+6	—14*
Blood pressure, systolic	114	112
Diastolic	46	43
Pulse	68	70
Respiration	21	24
Temperature	98.3	97.5
CO^2	42	44

In the respiratory metabolism data, significant data are again encountered. The younger boy is normal, the older exhibits a definite degree of hypofunction, even though the value obtained is above the true, as the patient was slightly restless

*Slightly restless; value too high.

and therefore not strictly basal. The physical findings show a practical equation; although the older boy's temperature is slightly low.

B-379.

Dist. vis., R. 10/10; near vis., R. J-1; R. pupil, size 5 mm.; shape, round.

Dist. vis., L. 10/10; near vis., L. J-1; L. pupil, size 5 mm.; shape, round.

R., pupillary reaction to light, nor.; consensual, nor.; accom. conv., nor.

L., pupillary reaction to light, nor.; consensual, nor.; accom. conv., nor.

Rotations normal. Convergence, normal. Media, R., clear; L., clear. Fundus, R., disc shows organized exudate, slight amount, covering some vessels; L., same condition, less marked.

Interpretation: Macular vision normal. Contraction of form and color fields. Evidence of previous inflammatory condition of nerveheads.

B-380.

Dist. vis., R. 10/10; near vis., R. J-1; R. pupil, size 4 mm.; shape, round.

Dist. vis., L. 10/10; near vis., L. J-1; L. pupil, size 4 mm.; shape, round.

R., pupillary reaction to light, mod.; consensual, mod.; accom. conv., nor.

L., pupillary reaction to light, mod.; consensual, mod.; accom. conv., nor.

Rotations, normal. Convergence, normal. Media, R., clear; L., clear. Fundus, R., same condition as left, but less marked; L., disc shows organized exudate, slight amount, covering some vessels.

Interpretation: Macular vision normal. Contracted form and color fields. Enlarged blind spots. Disc changes suggestive of previous inflammatory condition. Findings questioned slightly for accuracy, but in gross are reliable.

In the eye examinations, the original records of which are here included, we find in both cases certain residua of an exudative process which, in the examiner's opinion, may point to an earlier and unrecognized encephalitis or other inflamma-

tory condition. The other findings, however, show that the older boy departs far more from normalcy than does the younger. The lower mental level of the older boy does something, at least, to lessen the absolute value of these findings, but there can be no question that relative field contraction and blind spot enlargement are here presented.

B-379.

Percentage hearing loss:*

	64 C	128 C	256 C^1	512 C^2	1024 C^3	2048 C^4	4096 C^5	8192 C^6
Right ear.....	49	34	41	36	38	43	34	32
Left ear.....	25	22	27	36	34	34	38	27

Rinne test negative with prolonged bone conduction. Both drum membranes show normal appearances of the cone of light without retraction of the membranes. Both tubes patent. The nasal septum slightly deviated to the left. No marked obstruction to breathing. Very small tonsils, vault clear.

B-380.

Percentage hearing loss:†

	64 C	128 C	256 C^1	512 C^2	1024 C^3	2048 C^4	4096 C^5	8192 C^6
Right ear.....	57	45	45	52	57	54	56	D
Left ear.....	57	45	45	52	57	58	D	D

Rinne test positive with little change in bone conduction. Both drums show past discharge, although healed by scar tissue at this time. Both inferior turbinates slightly hypertrophied. Some tonsil remaining on both sides, no palpable cervical glands.

The two audiograms show appreciable loss in the conversational area, with the younger boy better in the lower tones than the brother. The older boy, however, was not cooperative. The functional tests are atypical in the older boy, while they show Bezold's triad in the younger. The cause of the positive Rinne in the elder is due to the past suppurative condition of

*The use of the audiometer is through the kindness of the New England Telephone and Telegraph Co.

†The use of the audiometer is through the kindness of the New England Telephone and Telegraph Co.

the middle ears. The X-ray report of the older showed a deep sella, 11 x 13 mm., as well as deep marking of the diploic veins and a calcified pineal gland. The static labyrinths of both boys were quite normal as was their past pointing.

Aside from their common parentage and heredity, these two boys present but two points in common. The first, their presenting symptom of deafness, and the second, the observation of exudate in the fundus examination, presumably indicative of an earlier inflammatory process. Concerning the etiology of this latter condition, both histories are uninformative, and one is forced to postulate the existence of some condition at an earlier date which was not recognized and recorded. Taking the picture of the younger boy for the moment, there is but little to suggest an endocrine disturbance. His normal basal rate, relatively normal physical findings, and the normalcy of the majority of the supplementary tests, practically eliminates an endocrine disturbance from the etiologic field. His suggestion of some kidney disturbance, the abnormality of the blood morphology, and the eye findings are suggestive of a pathology, it is true, but not one of endocrine origin. This case could be reported only as not demonstrably endocrine and the hearing failure traced to metabolic disturbances engendered by aberrations in which the ductless glands play no part.

With the older boy, on the other hand, two possibilities present themselves. If one takes the laboratory and clinical findings without context, we have a fairly well defined picture of a pituitary dysfunction. Salient points, such as the lowered basal rate with slight downward tendency of the concomitant physical findings, markedly depressed sugar tolerance, high blood uric acid and an otherwise normal blood, coupled with a definite eosinophilia, suggestive X-ray findings, although it must be emphasized that the experience of this laboratory shows no significance attaches to the calcified pineal, and the striking eye findings, are all frequently observed in the aberrations of the pituitary. The exudative residuum observed in the eye, however, duplicating that observed in the younger boy, raises a definite question. Data are lacking concerning the influence of lesions of the central nervous system on certain of the tests, but it has been shown in this laboratory that

a marked depression of the sugar tolerance and moderate fall in the basal rate are consistent with certain of these conditions. There is here then the possibility either that we are dealing with that type of pituitary dysfunction in which the anterior lobe is under- and the posterior lobe over- active, or that an inflammatory condition has injured the central nervous system and produced, on the one hand, the eye findings observed, and on the other, has determined the striking departures from the normal which under other conditions would be regarded as definitely indicative of a pituitary condition. A possible hypothesis uniting these two postulates would be that the pituitary has suffered in the damage caused by an earlier inflammatory process and thus influenced the laboratory picture by the superimposition of certain characteristic pituitary findings. A possible support for this might be found in the tendency to polyuria exhibited by the older boy. In the light of more recent work, this symptom is referable not to the pituitary per se, but to damage in the neighborhood of the pituitary, as has been shown by Houssay. The experimental exhibition of anterior lobe extract is permissible in this case, and it would be very interesting indeed to see how far this form of medication will influence the patient's several presenting symptoms.

These two cases are felt by the author to illustrate in an interesting manner the general thesis of the etiologic background of otosclerosis. In these two boys there is a common end result, namely, a progressive failure of hearing. In both, again, there are obvious disturbances of general metabolism. In one, the younger, there is no evidence of an endocrine factor in the condition. In the other, however, there are many observations conforming to the established indices of a pituitary disturbance. In many particulars the two boys are the antithesis of each other. In a few only do they coincide. If, as the writer believes, a factor in the determination of the condition known as otosclerosis is the disturbance of normal metabolic processes, we must inevitably have cases in which an endocrine element is present, as well as those in which it is absent. And when present, the endocrine influence exerts itself by a disturbance of the many metabolic equilibria with the ear condition determined by constitutional tendency.

The results of this paper can be summarized as follows:

SUMMARY.

1. It would seem to be established that many and widely divergent factors can excite the appearance of otosclerosis in a predisposed person.

2. The essential factor underlying the morbid change is possibly a chemical one affecting the nutritive stability of developing and fully developed bone and cartilage.

3. In a dominant percentage of cases the several endocrine glands are direct etiologic factors through their influence on normal metabolism.

4. In cases of otosclerosis without firm ankylosis of the footplate of the stapes or other irreversible organic changes, a response to the proper glandular treatment may be expected with reasonable confidence.

5. The constitutional tendency or hereditary influence would seem to be a dominant factor in determining the condition of otosclerosis where an endocrine or nonendocrine pathology produces disturbances of metabolism.

6. That the work so far done is to be regarded as suggestive rather than definitive of a possible ultimate solution of the highly complex problem of the etiology of otosclerosis.

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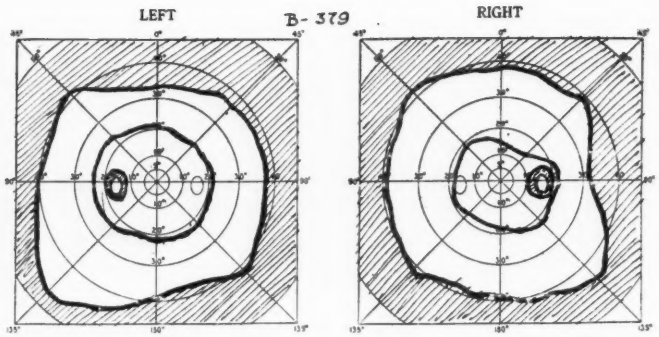

Fig. 1.

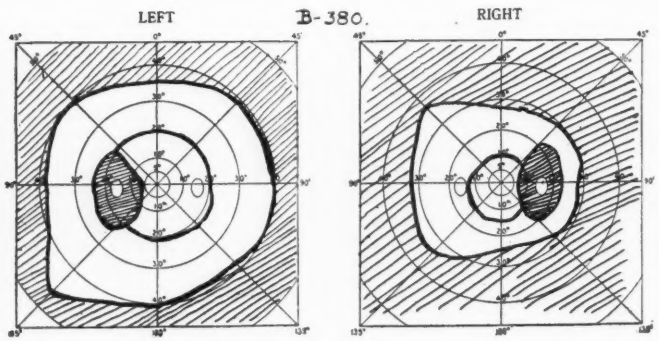

Fig. 2.

B-379.

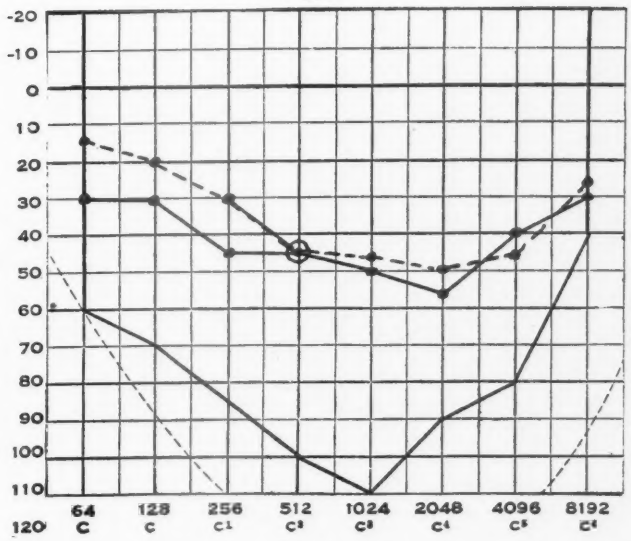

Fig. 3.

B-380.

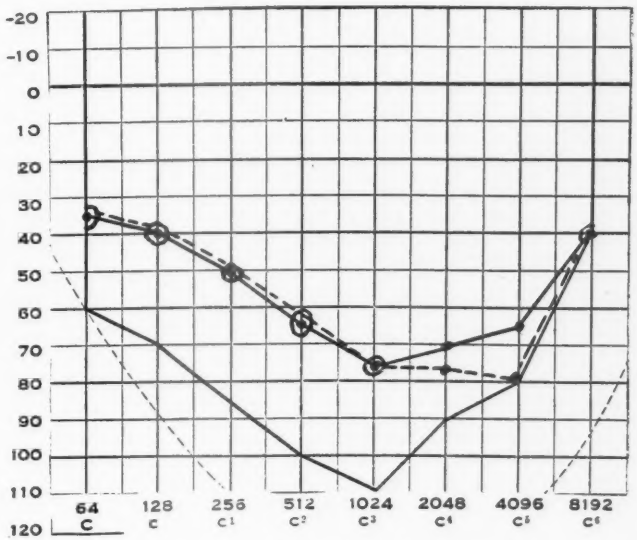

Fig. 4.

XLIII.

COLDS, INFECTIONS AND PSEUDOCOLDS.

By Wolff Freudenthal, M. D.,

New York.

In the foreword to his work on "Colds," Sticker says: "Colds! for a long time a meaningless word in the science which Leeuwenhook, Fahrenheit, Morgagni, Laennec, Fechner, Liebig, Roentgen have enriched with new ideas. Hot or cold foot bath for a cold? Probably an unimportant question in the art that has received new impetus from men like Morton, Lister, Faraday, Pasteur, Koch, Behring and Ehrlich. Coryza, cough, frost bite! Small affairs for a mind that once was allowed to stand on the pinnacle from which he overlooked the endless, ceaseless ebb and flow of pestilences so pernicious to entire nations." Small affairs, I should say, but of greater import than many a physician brought up in the light of modern bacteriology might dream. Has not Sydenham, a physician we must respect even today—has he not said that more people die of colds than of pestilence? Perhaps Sydenham did not know what a cold was. Bacteriology had not been invented yet. Nowadays, everybody, not only every physician, but every layman, seems to know what a cold is, and, by merely looking at the sufferer, will make the diagnosis in an instant. Certainly for such a diagnosis an intranasal examination is absolutely indispensable, something that it would seem superfluous to draw attention to today, more than sixty years after the medical world has learned to make an intranasal examination. Yet, as a matter of fact, statistics are compiled, papers are published on "colds" with thorough bacteriologic findings, but without any reference to clinical examinations of the nose and throat. When we notice that men of excellent standing describe all kinds of "colds" and "catarrhs" of the nose without mention of intranasal examination in a single instance, we cannot but recall the prelaryngoscopic era, the time when no less a man than George Washington suffered

and died of a laryngeal affection that was called by his physicians a "synanche," while in reality it was an abscess of the larynx,* which nowadays would have been cured in most instances. George Washington's physicians had no means of inspecting the larynx and nobody would blame them for the untimely death of their distinguished patient. But today there are means of examination and they ought to be utilized.

How easily one may be deceived is shown by the following case, which is one of many. A patient, butcher by occupation, consulted me complaining of a cold. He had it almost the year round, and every few weeks had a fresh attack. As he was a butcher and had to go into the refrigeration house frequently, it was not unnatural that he should have such colds. On examination, however, nasal polypi were found. This case serves as an example of many others which a writer has used to prove the infectious nature of "colds."

There are so many instances in which we can be deceived in the same way in our diagnosis that it seems fitting to divide my subject into three classes, viz.:

COLDS, INFECTIONS AND PSEUDOCOLDS.

I. Colds.—Many years back a well known physician remarked that it takes courage to write on colds. One feels the same way today, when everything is considered settled from a bacteriologic point of view and the clinical side of the question so greatly neglected. On the one hand there are men who say that a deleterious causative factor produced by a low temperature cannot be ignored. Although scientific, i. e., experimental, proof has not been brought forward, they are sure that intense cooling of the body may produce "catarrh" of the respiratory tract, even pneumonia, also kidney disease, rheumatism, etc. On the other hand, there are those who claim that it has not been demonstrated in a single disease, not even the ordinary coryza or cough (sic!) that it can be induced by simple exposure to cold, nor that it ever originated in that way.

A priori is it not intelligible that the physiologic functions of the body should manifest great fluctuations under the in-

*Wolff Freudenthal: "Unusual Instances of Laryngeal Abscess," The Laryngoscope, November, 1911.

fluence of cold weather in winter and intense summer heat? These oscillations must be in proportion to the outdoor temperature. They will often approximate more or less close to the borderline of pathology, but cannot be considered as yet a disease. Is it not an old experience that changes in temperature are associated with many minor and major ailments, especially of the respiratory system? It is well known that with the decrease in the outside temperature, an increase of the metabolism occurs, and that higher temperatures have the opposite effect. Do we not observe a host of affections of the respiratory organs just at the transition of periods from fall to winter and from winter to spring? Is not the intensity of these affections greater in cold than in warmer weather? The possibility of a cold as an etiologic factor had to be admitted even by Ruhemann, although he is one of the principal representatives of the infection theory. Furthermore, Wernich's idea that catching cold as a cause of disease cannot be considered seriously and that meteorologic factors are without any influence on morbility and mortality cannot be sustained by clinical experience. We simply are forced to believe that there must be such a thing as taking cold. How is that produced?

There is heat perception as well as heat regulation, and the organ that acts as an indicator of changes and warns of any danger, the organ that protects the body from loss of heat as well as from overheating is firstly and mainly the skin. The mucous membranes of the upper air tract are only auxiliary, though important means in this function.

One of the oldest theories is that of J. Rosenthal (Berliner Klin. Woch., 1872). According to him, when the heated body, with its enormously enlarged blood vessels is exposed to a lower temperature, not only a considerable amount of heat is withdrawn from the system, but the cooled off blood soon reaches the inner organs and cools these much more quickly than if the body had not been overheated before. In other words, the blood vessels of the skin become paralyzed by the heat, contract as soon as the temperature is lowered and cool even the blood of the inner organs.

One author, Alois Lode, cannot understand how according to this theory because of the chilling of a small area—the neck,

for example, the foot could be affected to such an extent that it would become painful or that the inner organs could be involved. Yet this mode of taking cold occurs quite frequently. Or take the opposite direction. After getting wet feet certain individuals will develop a coryza or a diarrhea. From such experience we have learned that various ways may lead to "colds." It is probable that a cold affects directly the exposed part, as, for example, in facial paralysis or hyperesthesia of the trigeminus with herpes, etc. Or the effect is produced reflexly, i. e., on remote portions of the body. This may take place via the nervous system—where the vasomotor nerves play a great role. Or the blood is affected and carries the noxious material to remote organs. Furthermore, when through the dilatation of the blood vessels the skin is greatly reddened and a large volume of blood is carried through it, as just shown, a severe cold sets in. This will then be more intense, when the vasomotor nerves, through lack of exercise or through paralysis, do not produce sufficient contraction of the cutaneous vessels, which otherwise would occur after exposure to cold.

The influences of temperature changes upon the skin and mucous membrane come to our perception and cause us to make provisions against the loss of heat as well as against overheating. Or they are imperceptible (moderate air currents, for example) and cause automatic heat regulation. This will go on so long as the person is well.

The sensation of being chilly does not necessarily imply a cold, nor does a lowered temperature of the skin indicate a "cold" of the inner organs. The temperature of the skin depends largely on that of the outer atmosphere, but the temperature of the inner organs is uniform so long as we are well. According to Vincent, by the way, the temperature of the uncovered parts of the skin changes pari passu with that of the outer air from 37.5° C. to 22° C. in extremely cold weather. According to Kunkel, it changes between 32° C. and 35° C.

Thus heat perception and body temperature as well as outside temperature are entirely different things and the sensa-

tion of being chilled is independent of the temperature. Heat perception is the important factor. It tells us whether a sojourn in a certain locality, be it the open air or a closed room, is comfortable or not, and whether it is accompanied by a sensation of heat or cold. In many, probably the majority of civilized people, there is a hypersensitiveness to temperatures, and that is always a disadvantage.

Parenthetically it may be stated here, that over a hundred years ago the first attempts were made to measure the dynamic quantity of sensible cold. Only lately have these been taken up again. It is known that the thermometer alone does not give exact information of these processes. More is required, such as the humidity of the air, the winds, etc. For such purposes Leonard Hill recommends an instrument called Katathermometer. C. Dorno of Davos devised another one, which he named the Davos frigorimeter, and which is designed to measure the degree of physiologic cooling off.

When imperceptible and temperate air currents do become factors in taking cold, they create a hyperemia of the mucous membranes. And now come the advocates of the infection theory and say that the bacteria already present on these membranes are thus stimulated to new growth and finally cause an infection. This is decidedly not in accord with the writer's viewpoint. It must be wrong, for a mere accumulation of normal blood can never produce an infection; in fact, we make use of it often in the socalled Bier's hyperemia treatment. In accordance with this conviction of mine is a statement by Strasser, who thoroughly had studied this question. According to him the amount of blood circulating in an infected area is of great significance. The more blood the greater the protection, the better the circulation and the easier the fight against bacteria. From this we learn that an organ is affected after exposure only when the blood supply is impaired with resulting weakened condition of the body cells; in other words, here as in other diseases the locus minoris resistentiæ is affected first.

Thermic stimuli exert a powerful influence on the blood vessels and the preservation of the body heat depends on the compensatory processes between the heat generation of

the organism and the outer temperature. If this adaptation fails, too much withdrawal of heat will take place and disease will result. The naked savage uses his skin for such purpose. That and his inner safety valves prevent changes of body temperature. Thus any human being may become acclimated in various localities. But if he seeks another climate he has to adopt artificial means for his protection, such as the civilized man unfortunately uses in hyperabundance, i. e., garments. As a rule, too much clothing is worn, and what happens? The skin that should have a temperature of from 5 to 15° C. lower than the inner organs becomes unduly heated. The warmer the skin, the more dilated are its capillaries. Thus much more blood comes in contact with the cold air, much more heat is given off and much more readily does taking cold occur. This is only too frequently observed after overheating of the body, when followed by exposure to cold air. Other factors, however, are of equally great importance. They are mostly of a meteorologic nature, as the direction of the wind, the amount of humidity in the air, rain, snow, etc. But here, perhaps more than elsewhere, the immense variation in susceptibility due to habits, occupation, etc., has to be considered, nor can it be overestimated. The more accustomed the skin becomes to the stimuli of certain temperature, the stronger will have to be the atmospheric influences to cause a contraction of the capillaries, or in that way produce a cold.

But now again come the "infectionists" claiming that this is all antiquated and theoretical deduction. This can be refuted by any experienced practitioner, who without prejudice will investigate his cases carefully. He will tell you that in many of his cases a causative relation between a cooling off of the body and a consecutive disease cannot be denied, and that by prophylactic measures he is able to help his patients materially. The "infectionists" do not seem to believe in such experience. They rather assert that any "catarrhal" affection, "catarrhs" of the nose, pharynx, larynx and trachea, which are commonly called "colds," are nothing but an infection pure and simple. A simple "catarrh" of the upper air tract is not conceivable and even impossible without the presence and propagation of bacteria. This led Kuehn to pro-

pound the theory of latent microbism. It is known, says Kuehn, that numerous bacilli, mostly innocuous ones, are constantly found on the mucous membranes of the nose and throat. Very true, Waldeyer's ring as well as the Schneiderian membrane of the nose protect the body against the invasion of foreign bodies, principally of course, against bacteria, thus acting as a natural filter. If these tissues work efficiently, then there can be no infection. If they are defective, the protection fails, the microorganisms will penetrate the mucous membranes, get into the inner organs and become pernicious. According to Kuehn, these bacteria are omnipresent and dormant, waiting for the moment when they are stimulated to new growth. Now he claims that exposure to draft, etc., awakens them, the protective walls against the invasion fall and the infection is there. The writer cannot help calling this idle theory, built on sandy soil. The experiments that were made to prove this—numerous, indeed—prove nothing.

Dogs and guinea pigs that had been exposed to intense local and general cold stimuli, showed hardly any organic alterations, while in other experiments animals that had been treated in the same way succumbed quicker than those that had not been exposed. Among others, Chodounsky, after many experiments on animals and his own person, denies absolutely the exclusive and sole infectiousness of a coryza or a cough. He jumped into icy water in February without any detriment to himself. But, says Kuehn, "if he had exposed himself during the night to a prolonged cold draft, an infection would have resulted positively." Why an infection then? When Kuehn and other writers claim that the cold air is a negligible factor and that the main thing is whether or not the atmosphere contains bacteria, I ask: Why should these germs show up in the night during a prolonged sleep? If there be less resistance during sleep, very well. Then we have to fall back on our theory of the influence of cold air, etc.

The advocates of infection claim that the north pole explorers, Nansen and Johansen, had exposed themselves to the coldest temperature possible, that Nansen too had jumped into icy water, and yet no cold resulted. Since according to Kuehn and others, an omnipresence of microorganism is to be found in every nose and throat, I ask: Did Nansen and all his men

have absolutely sterile conditions of the membranes of these organs that they were able to avoid infection? It is not likely that such was the case. But all this can be easily explained. Firstly.—Short exposure to intense cold has the opposite effect on the skin and mucous membranes, viz., a contraction of the blood vessels followed by a reactionary dilatation and a hyperemia which increases the power of resistance. This in itself may explain the fact that no harm resulted to Chodounsky nor to Nansen. But there are other proofs. Let us for a moment consider such diseases as pneumonia, rheumatism, etc. For many physicians the finding of a diplococcus in pneumonia is sufficient to make it an infectious disease. The diplococcus may be found, but there are various causes of the disease and a variety of pneumonia. To me it appears quite positive that exposure to cold, severe and unaccustomed cold, plays an important role in the etiology of this disease. Thus Zillessen (Inaug. Diss., 1899), to mention only a very few instances, cites the following case: A middle aged man fell into the river adjoining his house. For some reason he had to wait a long time during a cold night, wet all through, before some one opened the door. The next day pneumonia set in, of which he died. The autopsy showed a typical croupous pneumonia. Another case occurred lately when the fierce epidemic of diphtheria broke out in Alaska. A man, named . . . rushed to Nome in an open sled with antitoxin. For hours this brave man had been exposed to a dreadful blizzard. He caught pneumonia and succumbed to it. I ask: Could there be a more sterile atmosphere than the wilderness of Alaska? This was not a case of infection, "pure and simple," but one of cold, pure and simple; in other words, one of exposure for a prolonged period to extreme cold atmosphere. Such cases are not rare, and you will find them when you look for them. Nansen and others were exposed for only a short time, and the reaction following was strong enough in this vigorous and hardened man to enable him to withstand the disease. In view of so many similar facts, it is impossible to understand how Ruhemann, who wrote a very interesting book on this question, could say: As regards pneumonia, pleurisy, angina, laryngitis and bronchitis, bacterial infection as a cause of these dis-

eases has gained the victory over colds, and has, so to say, taken the inheritance of the doctrine of catching colds. Has it, indeed? Is this true of angina and laryngitis? It will be seen later on whether Ruhemann was correct or not. In his opinion these diseases develop only when there is a predisposition, and this predisposition is based upon the presence of pathogenic bacteria. It must be present or otherwise every one exposed to the same draft or the like would become infected. To this I would say decidedly no. The truth must be looked for elsewhere. The cause of catching colds lies in a deficiency of the circulatory system and of the physiologic activity of the skin. In the skin, according to Ponndorf, certain antibodies are formed that protect the body against the invasion of different kinds of bacteria. That is to say, at the time when a person is in a state of hypersensitiveness due to a "cold," the protective action of these antibodies ceases temporarily and the equilibrium between toxins and antitoxins is interrupted in favor of the cocci that circulate in the blood.

Of course, these are all mere theoretical deductions, to which even R. Geigel in his book, "Wetter und Klima" (Weather and Climate), cannot fully subscribe. Geigel, too, however, believes that two things are required to produce a cold, i. e., an infection plus some cooling off of the skin. By that, the influence of the skin, by which immunity is produced and antibodies formed, is interrupted or greatly reduced.

To Ruhemann and others the matter is closed as regards the bacterial origin of rheumatism. If the effect of cold were the principal factor in rheumatism, they ask why should a child of six to fourteen years, with its delicate skin, not be attacked more often than a man in middle life? My answer is: It is not the delicacy of the skin, but its physiologic activity that counts. And not only this but the great physical activity of children and finally the state of their general metabolism. Moreover, as children are apt to have only one-third or one-half of their surface protected by garments, this is an advantage, not a detriment. They can stand changes in temperature much easier than grown people. Their agility of movements keeps the blood in good circulation and their metabolism, as a rule, is much better than in persons more

advanced in years. But even in rheumatism there are a variety of causes that may produce the disease, among which infection also plays an important role.

The blood circulation has been mentioned as important. The life of an individual, it is said, depends upon the elasticity of his blood vessels. In a similar way the protection of the mucous membranes and other parts of the system also depends upon the elasticity of their blood vessels, that is, their ability to dilate according to requirements and transport the necessary blood supply to the exposed areas. If this does not occur and the vessels remain contracted, the result is a prolonged anemia, then an inflammatory enlargement, increase of secretion and, in other words, a coryza or cold.

Most of us, therefore, nowadays are compelled to believe in a certain form of humoral pathology, i. e., a system that seeks to explain many of the symptoms of disease by an altered chemical and, I would add, physical condition of the blood.

The writer cannot resist the temptation to quote a few cases from his practice. Mr. N. N., a strong, healthy man, writer by occupation, had been sitting all day long at his desk, occasionally also near an open window, writing. Towards evening he naturally felt fatigued and at 8 p. m. had a severe attack of coryza. Several weeks later the same gentleman made a trip on his bicycle about 25 miles, came home, breakfasted and sat all day long near the open window without experiencing any ill effects at night. The atmospheric and other conditions were the same as before. I quote this instance as an answer to a statement that reads about as follows: In favor of the bacterial and against the physical etiology of colds it has been said that colds happen in people, who for days or weeks have not left their room. This is just what I claim and is a strong confirmation of my contention. A room is the very place in which to take cold. This is a warning that I should like to impress on every practitioner. The man referred to above was sitting quietly all day and working with his brain. His circulation was below par and a prolonged though imperceptible draft was sufficient to produce a cold. Later on, when he did the same work after vigorous exercise, nothing happened.

Another patient of mine went out rowing one afternoon. He exercised quite energetically, and when he came home threw himself on his sofa near an open window and fell asleep, drenched with perspiration. Unfortunately it commenced to rain and storm and there was quite a drop in the temperature. He awoke after a long sleep and could hardly raise himself; he had lumbago. I again ask: Was this an infection or was it not rather a plain, every day cold? Instead of lumbago or diarrhea, one may see quite as often coryza. When we take into consideration such cases, it is incomprehensible how one can speak of extreme infectiousness of coryza, which is supposed to apply to every kind of coryza. As we shall see later there are various other forms of coryza that have nothing whatsoever to do with either an infection or exposure to cold. But in the ranks of the "infectionists" there is a good deal of dissension. Most of them have not at all a clear idea of what they are aiming at. Some of them believe that there is a small group of affections which may be due merely to influence of cold without the action of bacteria, as certain forms of laryngitis, facial palsy and some intestinal disorders. Others seem to have come to the conviction that cold is an additional, sometimes important, factor in the causation of infectious diseases and that these would be impossible without the former. Such assertions naturally impair the strength of their theories. But theory aside, for the physician it is most important to know whether or not he can protect his patients against these colds. This we shall try to answer now.

Before doing so, let me add here briefly that uric acid in some, although very small number of cases, may play a role in "colds." Some people wake up in the morning with a slight coryza, etc., but by eliminating the uric acid from their system the condition improves.

PREVENTION AND CURE OF COLDS.

Catching cold, as has already been shown, does not depend exclusively upon a lowered outside temperature, nor on the duration of the noxious influence, but also on the constitution, age, state of digestion and, most of all, on the condition of the circulatory apparatus. Most frequently there is

also present some other cause, fatigue, a lack of something, etc. To protect an individual against hypersusceptibility to refrigeratory influences would in many cases be tantamount to a careful selection of his parents (Sticker). Such weakness is undoubtedly inherited often, but much more frequently acquired by unhygienic habits or adopted as a matter of fashion or fad. The almost hopeless fight against all these conditions should nevertheless be the task of every practitioner. But first of all we, ourselves, must be sure of our ground.

Everywhere one notices the tendency of some people to frustrate the evil effects of a steadily advancing civilization by a return to a more natural way of living, to become hardened and thus prevent colds. This is being done by some people, but is it done correctly, or is it overdone? These questions have to be decided first. The importance of colds —termed by some writers euphemistically "idiosyncrasy"—has seldom been underestimated by the laity, but now it is almost the custom to overestimate it. The writer has tried as strongly as he could to establish the fact that there is such a thing as taking cold. Following his deductions it would be rational to make an attempt at prevention. But the fear of catching cold has assumed such dimensions that it terrorizes large masses of the population and is to be regarded not only as ridiculous but fraught with dangerous consequences. The manner in which we dress, how we live, when we go out or stay at home, everything is governed by one idea, the ghost of catching cold. Although it may be true that many individuals frequently catch cold in the open air, this is no valid reason why we should exclude ourselves from it. On the contrary, one should go out in the open air in all kinds of weather, since just those who do not get enough fresh air are apt to suffer most. Another prejudice that prevails here more than elsewhere is that one should go out little or not at all in rainy or snowy weather, that one is liable to get wet feet and thus risk his health. But why this should be more detrimental than wet or cold hands no one has been able as yet to explain to me.

True enough, in damp weather more heat is lost than in dry weather. But since the main supply of heat is produced

in the muscles, from where it is pumped into all parts of the system by the heart action, all one needs to do in damp weather is to put one's muscles into action and thus prevent too much cooling off.

Speaking from a purely bacteriologic point of view, such weather conditions ought to be preferable, since much less bacteria are found in the air during rain or snow than in dry weather (see results by F. Weltz and others). I would say, however, that in a large number of instances it is not a question of bad weather, but of good clothing. . .

When one "infectionist" says that we are as impotent to protect ourselves against taking cold as against influenza, etc., while another remarks that there is no hardening against the influences of temperature changes, we feel like advising these men to try hardening for a while, but try it in a rational manner.

IMPROPER CLOTHING.

Under this heading many authors decry the lack of sufficient clothing, while I believe, on the contrary, that many ailments originate from wearing too many garments and undergarments. Too many persons think that they have to put on winter clothes on the first of October or November and not discard them before the first of May. If this were really good common sense, the question arises: Are these heavy garments worn as a protection against cold weather or not? As soon as such a question is asked, one realizes the harmfulness of such customs. During these months there are temperatures as low as zero and as high as 65° F., perhaps occasionally higher. If one desires to protect his body, the protection must be different at zero from what it should be at 65°. Nobody can doubt that. Yet what happens actually? A gentleman of about thirty years of age, a man of education, called on me in February at a time when the outdoor temperature happened to be over 60° F. He was clad with a heavy overcoat, a sweater and heavy woolen undergarments. After my having drawn his attention to this he said that it was too early for a summer overcoat . . . At a medical meeting several women, all physicians, were sitting for hours

in the overheated room with their furs and hats on. Will they be susceptible to colds? But such habits are almost universal.

For these reasons it has been my custom to advise my patients to dress according to the temperature, and not according to the season. That should hold good at least for the climate in New York.

While we need some protection we should resort to some well thought out plan of hardening that will increase our power of resistance and will enable us to withstand without risk certain influences of weather. By such a system many of my patients have been kept free from "catarrhs" and "colds" for years, while others, often members of the same family, who did not believe in such procedures, suffered frequently, some even continuously as soon as colder weather set in.

As has been briefly remarked before, modern civilization forces the masses of the population into closed quarters, schools, factories, offices, etc., and into more sedentary occupations with the result that circulation is impaired and the heat regulation of the system suffers. Even in the country every child is forced to spend from 6 to 7 hours daily in a closed class room that prevents the exercise of his muscles. In the city it is worse. (Is education really of such value that the health of so many children should be sacrificed?)

In one of the newspapers there was a sketch of a proposed skyscraper, fifty stories high, which is to house all but two of the departments of the University of Pittsburgh. Saving of land space and reductions of upkeep costs were important factors in deciding on this type of a building. It will accommodate 12,000 students. As I understand it, everything will be in that building, including libraries, dining rooms, etc.

How was it possible, I ask, that such an abnormal structure could be thought of? Twelve thousand students to be housed under one roof. All day long they are prisoners in this building. They cannot or should not get out for a breath of fresh air—they would lose too much time. All day long artificial air and speed are handed to them. Saving, saving, saving in money and time. That is the watchword in business and is now the watchword of a university. The sacrifice of young

lives is overlooked entirely. Has no medical man in Pittsburgh been asked for his opinion? The whole idea is so horrid, so grotesque that the writer hates to think of it.

When the great German poet, Goethe, died, his last words were: "Mehr Licht" (More light). Some day some sensible person in Pittsburgh may cry out: More fresh air and more light.

UNDERWEAR.

It seems like an antiquated idea to speak against woolen underwear, yet on studying the subject I feel forced to do so. "Wool is a poor conductor of either heat or cold and is therefore always indicated." That is the way physicians often talk. But that is wrong. Most frequently one meets with highly warmed undergarments, the detriment of which is that it makes the skin extremely susceptible to any cold air, spoils the chemical regulation of heat and causes great loss of humidity. Worse than all this is, according to M. Rubner, the lack of ventilation. This is entirely due to too densely woven material worn on the body. This lack of ventilation finally leads to the deposition of a droplet like perspiration on the skin, causing the garments to become moist. The effect of this may be easily imagined, since the art of a rational raiment lies in the prevention of stagnating moisture on the body.

Winter underclothing—winter flannels; they seem synonymous, as a well known clinician of this city said (La Fetra). For so many generations has it been the rule, as mentioned above, to change the undergarments on say, November first, and to take them off on May first, that any discussion of the topic may seem superfluous if not heretical. Undoubtedly there are homes and climates in which such a practice is in strict accord with physiology and hygiene, but in our superheated houses and changeable climate this practice is a mistake, not only for the great majority of children, as La Fetra said, but for everyone.

TRAIN THE VASOMOTOR NERVES OF THE SKIN.

After experimenting many a year I must say that the majority of us are better off without any undergarments—woolen ones or not—and that applies to summer and the

greater part of winter. It is said that linen attracts and retains moisture. Well, suppose it does? If one perspires in summer, let him change his shirt and he will feel more comfortable. It makes a peculiar impression when one sees in a sanatorium near Dresden patients clad only in gymnastic trunks and shoes walking in the open air while the snow is falling. But it is seldom that any thing happens to them.

Even tuberculous patients with their reduced vitality stand the fresh air treatment, as is well known, wonderfully well. Is this not another warning not to wait until we get sick, but to make more use of the physiologic function of the skin while we are well? Do not destroy that physiologic function systematically, is my advice.

Man is born to go about naked. He received his skin, like any other animal, to protect him against the inclemency of the weather. The skin is a part of our respiratory apparatus, and a very important one. It serves to keep up a constant body temperature, which it helps to regulate. A suppression of the activity of the skin not infrequently leads to colds, etc.

History has shown over and over again that wild tribes were annihilated simply by putting on the clothes of civilized people. It is still the writer's opinion that the less clothes we can get along with, the better we are off. From that point of view the present style of women's dress should be the ideal one, if it represents any logical development of recognized hygienic laws. If one gets accustomed to wearing dresses reduced to a minimum, as compared with former years, it is all well and good. But if women wear furs in August and sit for hours in winter with hardly any clothes on or with fur coats in overheated rooms, the result will inevitably be the taking of cold in some part of the system.

The best protection against colds is our own blood. If the blood vessels will contract and then dilate, the blood will rush to the surface and serve as protection. In the warm blooded animal one of the most important functions, in all parts of the body, including the skin, is the irrigation, i. e., filling and refilling of the vessels with warm blood. As soon as one becomes accustomed to have his vasomotor nerves react quickly after an exposure in this manner, the skin will

stand most any refrigeratory insult without detriment. In other words, such a person has become hardened.

If muscles are brought into energetic action, will more antibodies be formed in the skin, as many believe? In all probability not. But waves of hot blood will be thrown into the circulation and act in a prophylatic way to prevent a cold. Consequently the main thing is to get your circulation in order. That can best be done by paying attention to exercises. The disregard of all exercise among many people is an underlying factor in many diseases, and not the least in catching colds. When one starts the day feeling chilly, it is not difficult to understand how quickly he will catch a cold at the slightest provocation. But if one exercises briskly early in the morning, he does not need to be wrapped up in heavy furs, etc. If the necessary heat is supplied from within, one gets along with less clothes. Needless to tell physicians that the struggle for life makes one physically stronger, especially when one has to run a few miles every day to save car fare and cannot afford luxurious, unhygienic garments. To see many young persons walk around in heavy furs, when the thermometer is way above the freezing point, is an insult to hygiene, a provocation to catching cold. As a rule, not every person has warm feet who wears thick woolen stockings with thick shoes, etc., but rather he who uses his feet more actively.

Now one need not walk nor do gymnastics all day long in order to keep up a good circulation. A certain well planned amount will suffice. The main factor in remaining well or rather keeping away from catching colds will always be this: Try to make good any deficiency in your circulatory system, and by all means train the vasomotor nerves of your skin well.

Since the majority of the population are kept in closed quarters the greatest portion of the day, theoretically the same garments should do for winter as well as for summer. Indoors there is artificial heat enough for protection. Outside one wears overgarments more than enough for the same purpose. Excessive covering of the body is a greater error than insufficient clothing. The more garments one gets accustomed to, the more he will need. They grow on a person

in the same way an intoxicating drug grows. We live in an era of intoxicating garments.

I cannot refrain quoting an old, long forgotten writer, Samuel Sheldon Fitch, who said in 1847 much that holds good today: "Great effeminacy of the constitution, and consequent tendency to consumption, is often induced by our treatment of the surface of the frame. Too much clothing greatly inclines to debilitate the system and lead to effeminacy. Suppose a person were to put his hand into a poultice and keep that poultice on for weeks; on removing the poultice the hand would feel as if it had no skin upon it; so sensible would it be to the external air. Too much clothing has the same effect. It greatly effeminates the system, and makes it so delicate that it cannot bear the least exposure whatever. Each successive addition of our clothing, beyond the point of health, instead of destroying the influence of cold, makes us more susceptible to its injurious effects, and infinitely more liable to those diseases that proceed from change of temperature."

But there are no set rules for health and longevity which will apply to the crowd. Every man is a physiologic and psychologic law unto himself (Howard). A great deal is done in this country for the mental development of the masses (too much?), but not enough for the physical training. It seems to the writer that every human being ought to do some active physical work, according to the state of his health. The best way for most people to take a rest cure would be a working cure of four to six weeks annually. Any kind of agricultural work or gardening, etc., could be done for the state. Instruction should begin in school. How much better would it be to prepare youth for agricultural work than for the military service!

If we strive to get rid of the hypersensitiveness of the skin, many affections, call them by whatever name you want, will then be avoided.

In hardening oneself one should not forget tepid baths, cold baths and cold showers. But if one believes that water is the sole therapeutic agent here he is mistaken. A child is not born into the water, it is born into the air. Conse-

quently the essential feature should be to get in contact directly with the fresh, invigorating air. The beneficial air can be utilized for hours, while a bath at best will last only a comparatively short time.

There still are practitioners who do not believe in hardening, nor that it has anything to do with the prevention of disease. Thus Geigel in all seriousness asks: Is what we call hardening perhaps nothing else but the selection of the fittest, or of those imbued with a great power of resistance? As an example he cites the case of a woman, who had given birth to twenty-three living children. In spite of hardening them carefully, she was not able to raise more than five. The writer could give hundreds of examples from his own practice in which hardening proved to be a great success. But is it not to be expected that twenty-three children born of one mother have less power of resistance than others, and that by other laws the natural selection of the fittest was made in this instance? The writer strongly believes in hardening and has had no reason to change his opinion during the many years of his practice.

If one overdoes the hardening process by wearing too few garments, it will by no means be as harmful as overdoing it the other way. A slight cold may set in, but that can be cured very quickly. Any of the coal tar preparations with a goodly portion of whiskey will cure most of these cases in less than no time. The less accustomed a person is to alcohol the better effect it will have.

Finally, if I may be permitted to give advice, it is to see to it, not so much that our patients become hardened, but more so that they do not get too soft.

II.—INFECTION.

The nose and throat are the avenues through which practically all the elements which nourish and sustain the body are taken into the human organism. They constitute the body's first line of defense against the invasion of disease. Nature, with marvelous foresight, seems to have anticipated that man in his multifarious occupations could be liable to breathe all sorts of impure and germ laden air, and to provide against the dangers incident to this, has supplied the nose and throat with a most complete and effi-

cient protective apparatus (Stuart Low). Every medical man knows that the Schneiderian membrane and Waldeyer's ring are a wonderful protection. Yet in about ninety per cent. of all cases of angina, for example, streptococci are found, and we ask: Is there any connection between these and the angina? In answering this question the problem is not the virulency or nonvirulency of the streptococci, but the important role the patient himself plays, as Waldapfel of Hajek's clinic pointed out, that is, his habits, his susceptibility to noxious influences, his general condition, etc. (Streptococcus hemolyticus, of course, is not present in normal throats.) Occasionally, when the virulency is too strong, the defenses will fail, allowing the enemy to invade the inner fortifications. In other words, bacteria will enter the system and then there is an infection. Practically all of the acute infectious diseases of childhood, and for that matter of grown people, begin as nose and throat troubles. In measles, scarlet fever, diphtheria, unmistakable signs of this are present. Even infantile paralysis, and as the writer has claimed long ago, tuberculosis* are now recognized by many authors as having their origin in the vault of the pharynx. The same holds good for influenza that so often starts with a coryza. These cases are very infectious, and every kind of precaution possible should be taken; the patients should be isolated, etc., and everything done to prevent the spread of the infection.

The wonderful work that has been done by the Boards of Health, especially by that of New York City, cannot be praised too highly. It is known all over the world. The writer has long since joined the army of those who admire the progress bacteriology has made in this country. At the same time, however, he desires to lay stress on the fact that not every ill or ailment is a bacteriologic infection, and that many of them can be cured or prevented by looking at them from a different angle.

III.—PSEUDOCOLDS.

An acute inflammation of the mucous membranes of the nose and throat, followed by obstruction and then by a

*W. Freudenthal: "Contributions to the Etiology of Pulmonary Tuberculosis." Annals of Otology, February, 1897.

diffuse discharge from the nose and eyes, is generally called a "cold." As these symptoms are often the same in affections which have no connection with exposure to a low temperature nor with an infection, I like to call them pseudocolds for lack of a better name. We have to differentiate here between affections caused by dust and those caused by certain other physical conditions in our atmosphere.

A. Dust.—Cosmic dust and clean dust from the virgin forest or plain do no harm, save as mechanical irritants. Can anybody imagine that there ever could be an infection from the wind and dust in the desert plateau of Thibet sweeping down from the Himalayas some eighteen thousand feet high? Or over in our "American desert," could there be a dust infection in those prairies? Certainly not. It is different in our large cities, which so often are not kept clean. Yet it is not infection I am speaking of here. Let us hear what Guthrie (N. Y. Medical Journal) has to say on this point.

"Human beings, more so than the lower animals, prefer to herd together. The savage moves about more, but is still herding. The civilized gentleman herds and rests at the same time, which may answer the following questions: Why . . . . does he hide his skin, that organ of respiration, from God's oxygen with tight clothing? Why does he war with his fellowman and murder his brother for greed and selfish motives, instigated by those not on the firing line? Why does he make the reproduction of his species somewhat of a vulgar joke? . . . As the world progresses more artificiality generates in the activities of our environment. Nature must be forced to keep up the speed mania in all our undertakings. So, as we are carried along in the flood of human advancement, let us consider any topic that may soothe our fevered career. The prevention of the production of active and disturbed unclean pulverized matter is an important item for serious consideration. I refer principally to the awful disturbance of poisonous dust, by the heavy and rapid traffic on our streets and roadways. These enormous monsters in tonnage may, perhaps, by jarring buried pipes, loosen up joints and liberate gases and liquids further to poison our atmosphere."

How much dust there is in every large city can easily be determined by placing a glass plate on the window. A few hours later you will find it covered with dust. Now, when there is a constant, never ending stream of automobiles, with their poisonous gases and impregnation with city dust, some of the best sections of large cities, though the most expensive, are at the same time the most unhygienic ones.

All this dust and whatever it may contain, stirred up by the immense traffic and not the least by the street cleaning department, is inhaled and gives rise to a cough or an acute coryza with attacks of sneezing, etc. Such cases are not due to refrigeratory influences or, in most instances, to an infection, but merely to a mechanical irritation. They disappear almost as soon as the nose and throat are cleaned out—i. e., the foreign bodies removed. An infection acts differently.

That constant inhalation of various kinds of dust reduces the body's power of resistance greatly is well known. Thus, for example, the mortality of pulmonary phthisis in the granite cutting districts of Vermont is very high. Is that due to any tubercle bacilli being present in the granite dust? Certainly not. The infection is a secondary symptom and appears at a much later period.

For all these reasons we have termed the symptom complex spoken of here pseudocolds. As with other conditions, the symptoms become more pronounced, the mucous membranes of the larynx, trachea and even bronchi become affected, the longer the irritation lasts. In that way a laryngitis, tracheitis or bronchitis may develop. For these reasons, putting such a patient to bed, keeping him or her for weeks in one room with "even" temperature, is an error in judgment. Such patients will improve in spite of the treatment, but in consequence they acquire a weakened constitution and often enough become semiinvalids all their lives.

Of much less importance than the dust is the use of cosmetics by millions of women nowadays. In some instances symptoms were produced that simulated a "cold" or hay fever. But since that question will be discussed at length in another paper to be published soon, suffice it to say that even in affections of the mucous membranes the detrimental use of

cosmetics should not be overlooked. These affections, too, simulate a "cold." This only apropos.

B. RHINITIS SICCA AND ATROPHICANS.

There are other factors in the atmosphere producing so-called catarrhal conditions, among which the lack of humidity in the air should, above all, be mentioned. For many years the writer has attempted to establish the fact that the people in New York City, although surrounded by water on all sides, suffer from a lack of humidity in winter. I have frequently measured the relative humidity in private dwellings, hospitals, theaters, etc., and have found that in cold weather it never reached the minimum requirement—i. e., 40 per cent of the point of saturation. Even in the best equipped places it usually came down to 30 per cent, 25 per cent and less.

With our centralized system of heating, the air is generally heated in the cellar and at the same time robbed of most, if not all, of its moisture. In this condition it is transmitted to our rooms, where it absorbs all the moisture there present. The result is that we inhale air whose humidity is abnormally reduced. The influence which this abnormally dry air has on the mucous membrane of the nose, throat, etc., is very deleterious. It is proved that dry air, if inhaled, will become saturated in the nose and nasopharynx—that is to say, the drier the air we inhale the more moisture will be given off by the nose and nasopharynx. The work of these organs will be so much greater with every particle of air we inhale. How long the mucous membrane of these parts will be able to furnish the necessary water supply will only, therefore, be a question of time. Consequently, we will, sooner or later, reach that point where these mucous membranes will be as dry as a well in summer. This will occur, for example, in very cold weather only temporarily in perfectly healthy persons, or permanently, as we see it in so many thousands of cases, in pathologic conditions.

Thus a dry rhinitis develops, and I could experimentally produce the same conditions in anyone, under like surroundings. Since the writer has a "sterilizer" going in his office a great deal of steam is generated, the walls are wet from hu-

midity, but he never finds too much humidity in the room for his respiratory organs.

There are two lessons we should learn from this. Firstly, in every hall, every room where there are many people, especially in every schoolroom, there should be not alone a thermometer but a hygrometer as well. A thermometer alone means nothing, according to our present knowledge of the physiologic functions of the body, while combined with a hygrometer it will mean not only comfort but the well being of many.

A hygrometer indicating a low percentage of moisture would not be of much benefit. For that reason the second requirement should be fulfilled, viz., to provide abundant steam to reach, at least, the lowest point required. In that way we will, so to speak, "sterilize" the respiratory organs of many persons, including school children, or, in other words, make them sterile or immune against many a germ that thrives better on dry soil. We shall spare the teachers and pupils many "colds," many a headache and a score of other preventable affections.

The old and antiquated hot air furnace, as well as steam heating, have to be abandoned, as they produce not only a lack of humidity, but in addition bring into the house ashes, dust, coal gas, etc. Sooner or later they will have to be replaced by a simpler and better method. Whether that will be by means of kerosene or gas, or by other means, cannot be said at present. Will you stop for a moment and consider the enormous advantages to be thus derived? No ashes, no coal gas, no dust in the house, no furnace man and no annoyance through the furnace itself (breaking down, repairing, etc.). And as for the city itself, the benefit will be equally great. The saving in the matter of cartage of ashes in a city such as New York would be enormous. No matter how strong a wind blows, the ash barrels are dumped into the unprotected carts and the greater part of the ashes blown into the faces of helpless citizens. I would suggest to every physician to inspect, if only for a moment, one of our public dumping grounds for ashes where the carts are emptied. On witnessing such a sight, one is not favorably impressed with the hygienic prog-

ress of our cities. But why the necessity at all of bringing coal to cities? The following excellent plan had been devised by somebody, whose name I do not recall. The coal is burned right at the mine, turned into electric light and power, and transmitted to the city, where it may be installed in every residence and sold like any other electric current. This would be the ideal, the cleanest, cheapest and most hygienic heating system possible. But I fear I am thinking fifty years ahead. Today there are too many who would oppose such a plan for purely mercenary reasons. Why should not our medical societies have a word to say about this question?

Returning to our main subject, we have seen that from the lack of humidity a rhinitis sicca may and does frequently develop. From such a condition an atrophic rhinitis and pharyngitis gradually result, with crust formations in the nose, in the throat and in the larynx. The harm done by these crusts can be seen every day by any laryngologist. When imbedded in the pharynx or larynx, they only too often cause a cough for which many a patient is sent to bed instead of into the fresh air. The cold weather has nothing to do directly with such cough or sneezing. It is solely the lack of humidity. No matter what kind of a mild medicament you may apply to the upper air tract, if you will only free it of all crusts and the tenacious mucus in the posterior pharynx, the patients will stop coughing, sometimes immediately. Thus after removal of the foreign material all the symptoms of a "cold" subside without further trouble.

Once an atrophic rhinitis has been established, the function of the nose will change and other symptoms set in. Thus when a person afflicted with atrophic rhinitis goes out into the open air, in warm or cold weather, and walks or does any kind of physical work, his metabolism is increased, and, pari passu, the secretion from the nasal glands as well. Under normal circumstances this secretion will be taken care of by the ciliated epithelium. Since this is destroyed in atrophic rhinitis, the secretion runs out from the nostrils and is apt to be mistaken for a "cold." In a similar way one will notice in bald headed people that when they perspire beads collect on the bald spots. In others, with a full head of hair, the secretion does not col-

lect, but clings to the hair, just as the rain is held by the leaves of a tree for future use.

In spite of all that has been said here, some colleagues will tell us that every coryza and every laryngitis is an infection, though no sign of it is present, or, for that matter, any trace of tuberculosis, a condition so often suspected by those who do not take the trouble to examine the upper air tract.

One fact remains, and that is this: Through these catarrhal affections in the nose and nasopharynx, other conditions may develop, some of which are simple, while others are of a more serious type. Thus in children adenoid vegetations, and perhaps tonsils too, are stimulated to new growth by a persistent nasal catarrh. And it does not matter whether this "catarrh" is due to a cold or other causes. Adenoids after operations will often grow again. They cannot always be removed "radically," because the entire mucous membrane cannot and should not be cut out. (It is somewhat different with tonsils.) For that reason one has to agree with Reginald F. E. Austin, that operations per se cannot and do not cure—they merely remove the effects of a cause, a product, so to speak. Consequently, the trouble for which an operation was performed frequently recurs. "Naturam expellas furca, tamen usque recurret." You may drive out Nature with a pitchfork, yet will she always return.

C. OTHER NASAL CONDITIONS.

Not only are the symptoms of a pseudocold produced by these catarrh conditions, with or without the formation of crusts, but any foreign substance that gets into any part of the upper air tract may give rise to cough or sneezing, or both.

A cough or sneezing is identified by the laity as a "cold." But cough, for example, may originate in any part of the air passages and from many causes. The writer gets a pharyngeal cough whenever his rooms have to be heated a great deal, due to the low temperature outside. If such a cough lasts a certain time, a laryngitis and rhinitis may develop. A few days in the open air in rainy, snowy or any kind of weather will generally restore a normal condition. But instead, thousands of these sufferers are put to bed, just because they have such a cough or such a "bad cold." A cough, by the way, may reflexly

result from the external canal of the ear by the irritation of the nervus auricularis vagi.

In a similar way, sneezing is caused by an irritation of the ethmoid nerves. Any irritation may do that, as different chemicals, foreign bodies, pollen, etc. It is for the physician to differentiate between a coryza brought on by these causes or by cold air, and this is not a difficult matter, as a rule.

To this category belong all sinus affections that discharge pus into the nasal cavity or into the posterior pharynx. The presence of nasal polypi often allows the nasal secretion to stagnate, causing the same effect.

Undoubtedly there are numerous other causes in the nose and throat that manifest themselves by the same symptoms. It is only our purpose to demonstrate the impossibility of diagnosing persistent or recurrent "colds" from the patient's own history without a thorough examination of the upper air passages.

If the harmful influences discussed here persist, if crusts and scabs and pus are allowed to stagnate for any length of time, the irritation alone will result in acute rhinitis, pharyngitis or laryngitis. To isolate such patients is absurd, but it is advocated and done. In this as in other fields of medicine, technical skill cannot do the work alone; clinical findings and clinical experience are just as essential today as they have always been. In a case of accessory sinus affection we do not rely on the roentgenograms alone, valuable as they are. The diagnosis must be confirmed by clinical observations.

In the foregoing the writer has given some of the ideas that have occupied his mind for many years. He sincerely hopes that the plans suggested in this paper may soon be materialized and is confident that only good can result from their adoption.

In the preparation of this article, the author has availed himself of several excellent publications pertaining to the subject, as for examples: J. Ruhemann: 1st Erkaeltung eine Krankheitsursache. A. Kuehn: Abhaertung und Erkaeltung. Georg Sticker: Erkaeltungskrankheiten. Berlin, 1916, and others.

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XLIV.

THE ROLE OF CILIATED EPITHELIUM IN SINUSITIS.*

By Frank L. Phillips, M. D.,

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The study of the following anatomic descriptions and experimental observations was prompted by the statement: "When there is stasis of fluid mucus in the nose it is due to paralysis of the epithelium, the cilia being paralyzed by the toxins of the organisms present."

ANATOMIC.

The mucous membrane of the nasal cavity is divided into three more or less distinct regions: that of the vestibule, the nasal fossa and the paranasal sinuses.

The Nasal Vestibule.—As a sort of antechamber to the nasal fossa we have the nasal vestibule, which corresponds very closely in its extent to the cartilaginous nasal wall. At the nostril the skin of the upper lip and wing of the nose passes into the vestibule and for a distance retains its surface characteristics of a flat stratified epithelium with superficial horny cells. The skin characteristics, however, are lost about the middle of the vestibule, changing here to stratified flat epithelium with an absent horny layer, resting on an underlying connective tissue propria. The epithelium of the fore part of the vestibule rests on a connective tissue corium continuous with that of the skin.

The Nasal Fossa.—Conforming with the double function of the nasal cavity proper there are two areas of the lining mucous membrane (Schneiderian) that differ in structure, the respiratory portion and the olfactory portion.

The Respiratory Portion.—The stratified flat epithelium of the nasal vestibule gradually assumes the characteristics of

*Candidate's thesis presented before the American Laryngological, Rhinological and Otological Society.

the respiratory mucous membrane—that is, a stratified ciliated cylindrical epithelium; the nasal respiratory mucous membrane varies in thickness. Over the inferior turbinate, a portion of the middle turbinate and adjacent portion of the septum the membrane frequently reaches a thickness of several millimeters; elsewhere it may be considerably less than a millimeter. The membrane is intimately attached to the periosteum and perichondrium of the nasal fossa. The stratified ciliated cylindrical epithelium of the nasal respiratory mucosa rests upon a cribriform basement membrane. Ciliated cells extend the entire thickness of the epithelium; the slender ends, being deeply placed, resting upon the basal membrane, and the more bulky ends with the attached cilia forming the free surface. The cilia are least developed over the superior and supreme turbinates. They attain a length of from five to seven microns and produce a definite and characteristic current toward the choanæ.

The Olfactory Portion.—The olfactory portion occupies a small portion of the nasal fossa, being confined to the cephalic third of the nasal septum, nearly the whole of the superior turbinate, and a very small portion of the middle turbinate. It is demarked from the respiratory mucosa by its yellowish color and greater number of nuclei.

The olfactory mucosa consists of a surface neuroepithelium with a subjacent tunica propria. A definite and distinct basal membrane is wanting. The neuroepithelium is composed of specific sensory (olfactory) cells, sustentacular (supporting) and basal cells.

The sustentacular cells are tall, columnar, nonciliated epithelial cells and are considerably broader than the olfactory. The deep ends of the supporting cells often terminate by branching into two or more processes, in the intervals of which are found basal cells, which are small, flattened pyramidal elements that form the deepest nuclear zone of the olfactory neuroepithelial layers.

The olfactory cells are bipolar, fusiform and contain spherical nuclei located between the deeper parts of the supporting cells. The cells extend through the entire thickness of the neuroepithelium. Their peripheral processes are short and pass to the surface of the mucosa through openings in the olfactory

limiting membrane, each process giving rise to a hemispheric vesicle, which in turn terminates in six to eight minute hair-like processes of fine cilia—the olfactory hairs.

The Paranasal Sinuses.—The paranasal sinuses are lined by mucous membrane directly continuous with that of the nasal fossa, which it resembles, except that it is much thinner and contains fewer glands. Moreover, it does not assume the characteristics of an erectile tissue.

The mucosa of the paranasal sinuses and cells is composed of a stratified ciliated columnar epithelium, invaded by numerous lymphoid elements, resting on a very delicate basal membrane and tunica propria. This tunica propria is firmly adherent to the underlying periosteum, especially so in the frontal and maxillary sinuses, less in the ethmoid labyrinth and still less in the sphenoid sinus. Unlike the mucous membrane of the nasal fossæ, that of the paranasal sinuses is poorly supplied with elastic fibers. Moreover, the glands, mucous in type, are few and scattered as compared with the glands of the nasal cavity proper. In the maxillary and sphenoid sinuses the glands are most plentiful in the vicinity of the ostia of the cavities.

In spite of the extreme delicacy and thinness of the mucous membrane of the paranasal sinuses and its firm adherence to the periosteum, it is readily influenced and greatly thickened by pathologic processes. It is particularly prone to thickening, rapidly so in vicinity of the ostia of the maxillary and sphenoid sinuses, owing to the greater looseness of structures at these points.

The current produced by the cilia of the epithelium of the paranasal sinuses is toward their respective ostia or apertures of communication with the nasal fossa. The current produced by the cilia of the epithelium of the nasal fossa is toward the choana.

A. L. Yates, writing on "Methods of Estimating the Activity of the Ciliary Epithelium Within the Sinuses," states that by using lampblack as an indicator, he observed that by the action of the ciliated epithelium the mucus was rolled over and over, thus enveloping all the foreign particles. Using .4 per cent indigo carmin in normal salt solution at body temperature, he found that the colored fluid appeared to follow a definite

path, as follows: It passed along the upper part of the middle meatus, then outwards and downwards, eventually following a definite track in front of the eustachian tube. From this situation it passed downwards between the attachment of the soft palate and the posterior wall of the pharynx, and continuing passed as a definite stream which lay just behind the posterior pillar of the fauces. But from this part of the stream some of the fluid passed in all cases anterior to the posterior pillar of the fauces, and found its way into the recess between the tonsil and the posterior pillar.

At the level of the tongue the main stream divided into a larger and smaller portion. The smaller portion was conveyed across the dorsum of the tongue in the position of the lingual tonsil, and from thence in some cases it passed into the glotto-epiglottidean pouch, and in other cases was swallowed directly. The larger portion continued outwards along the posterior margin of the pillar of the fauces, and thus reached the sinus pyriformis and continued down this to the posterior aspect of the arytenoids, finding its way into the interarytenoid space.

In one abnormal case, where the mucus was stained and where its progress was slow, it found its way into the trachea through the interarytenoid space. The bulk of it remained in position for a short time behind the arytenoids and was then swallowed.

When placed in the posterior ethmoid sinus the fluid passed into the superior meatus, and escaped from this into the nasopharynx and was conveyed outwards for a short distance until it occupied a position at the upper part of the nasopharynx, immediately in front of the uppermost part of the eustachian tube. Here it divided into two streams, a larger, which passed downwards and joined the position of the stream from the antrum, and a smaller, which passed over the eustachian tube and then coursed downwards through the fossa of Rosenmüller and then curving forward, joined the antral stream at the lower border of the soft palate.

If the fluid was injected into the sphenoid sinus it passed from the opening of the sinus in a downward direction until it reached the highest point of the posterior choanæ. Here it turned backwards, and, spreading out like a fan upon the vault of the nasopharynx, was conveyed in a series of streams,

the outer of which made its way into the lower part of the fossa of Rosenmüller. The inner stream passed in an outward direction until they joined together in the position of the antral stream, about one-half inch below the lower margin of the soft palate, and the further course of the fluid was as that described for the antrum.

The ciliary action in cases of sinusitis was somewhat different. When mucus or pus is present in any of the sinuses the rapidity of the flow of the indigo carmin solution may be greatly modified. In cases where the contents of the sinus are mucus only, the rate of flow is little altered. In cases where thick gelatinous pus is present in the sinus, there is some slowing of the stream, but this is slight. In both the above cases the sinus contents are not stained by the fluid which is injected, and on washing out the sinus the condition generally does not tend to recur.

In cases where the sinus contents consist of thin green pus two noticeable alterations are seen. The pus stains blue, but not as a rule deeply. The fluid is conveyed away along the track from the sinus with considerable sluggishness, and strays from this, and tends to pass over the areas where contrary ciliary streams meet, as for example, at the edges of the eustachian cushion or at the posterior part of the larynx. In well marked cases the mucous membrane appears to stain, but it is found that this stain can be wiped off and consists of a thick layer of mucopus.

In cases of sinus infection of the atrophic type, no pus is found within the sinus, but the fluid contains microorganisms and débris in some quantity. The fluid is conveyed away very sluggishly, and there may be complete stasis of the fluid within the sinus, and the part of the track nearest to the sinus may appear to be deeply stained, and this stain may remain in position for some hours unless washed away by douching. In two cases in which fifteen cubic centimeters of a .4 per cent indigo carmin solution were injected into the antrum, and where this marked stasis was present, indigo carmin appeared in the urine in three-quarters of an hour, and a similar quantity of indigo carmin swallowed at a subsequent visit did not appear in the urine at all.

CLINICAL ASPECT.

Cases in which the mucus was unstained showed only local symptoms. Cases in which the mucus was stained and ciliary paralysis was partial showed remote local symptoms, such as laryngitis. Cases in which the cilia were apparently totally paralyzed and the secretion of mucus was absent and microorganisms present showed in some cases remote symptoms, such as arthritis, but in well marked cases of this type of sinus infection with atrophy, signs of sinusitis, as revealed by ordinary methods, were scanty.

The ciliary activity may be estimated more simply by using liquid paraffin to wash out a sinus on one occasion, and saline on the next, when the amount of liquid paraffin remaining in a sinus forms some measure of the ciliary activity.

CONCLUSIONS.

It would appear, therefore, that the severity of a sinusitis or sinus infection depends upon the power of the mucus to prevent its destruction by the inspired microorganisms, and if the property that mucus possesses of preventing the passage of water, and hence the water soluble toxins, is destroyed, that a progressively increasing poisoning by the toxins of the ciliated epithelium occurs, and that, as this ciliary paralysis increases in amount the secretion of mucus is also paralyzed, until finally the microorganisms come to dwell in a symbiotic state in the sinus, and the toxins from them are readily absorbed with the production of remote symptoms.

Secondly, that the intimate relation of the tracks from the sinuses to the eustachian tube suggests that eustachian catarrh is a secondary manifestation of sinusitis, and that the intimate relation of the tracks from the sinuses to the tonsil suggests that the initial enlargement of this is frequently due to sinusitis, although the tonsillar enlargement may, of course, be due to other causes.

In discussing ciliary motion, Verworn,[3] in his General Physiology, states that in contrast to most forms of muscular motion, ciliary motion is automatic—i. e., the impulses that lead to it arise in the cilia themselves. It has been determined by experimentation that the cause of the motion is seated in the

protoplasm of the cell body, for if the isolated cilia possess absolutely no protoplasm at their bases they are wholly motionless. Most cases of ciliary motion are distinguished by their rhythm. The individual cilia of a row, beginning at one end, contract in exactly the same rhythm and succession, so that every beat of the first cilium is followed by a beat of the second, then of the third, and so on. In this manner there occurs an extremely delicate and regular play of the cilia, which gives the impression of wind sweeping over a field of grain.

Englemann, in 1879, demonstrated that acids, alkalies and salts, carbonic acid and various alkaloids have like effects, which always consist in an augmentation of the activity of the cilia, the rate of their beat being considerably increased. Ciliary motion is likewise augmented by increasing temperature up to a certain degree, and also by the galvanic current. Also in 1868 Englemann found that the vapor of ether or chloroform caused a depressing action upon ciliary motion, as did also quinin and chloral hydrate. Following the technic described by Yates, various cases of sinusitis, frontal and maxillary, have been studied and the findings have been essentially as reported by him, as to the ciliary action and the pathways followed by the stains.

The frontal sinuses of large dogs were laid open, and in one sinus a minute quantity of India ink was placed on the floor of the sinus. The ink was picked up by the cilia and moved for a short distance up the posterior wall toward the roof of the sinus. It then turned at almost a right angle and was propelled forward and downward toward the frontonasal duct, through which it finally disappeared. It was interesting to note the rapidity with which the cilia rid the sinus of the ink, it being perfectly easy to note the advancing line of the stain, and also that as the ink was carried along no trace of it remained to stain the mucous membrane. In forty-five minutes the advancing line had traveled to the frontonasal duct, and in two hours all the stain had disappeared.

The frontonasal duct of the opposite sinus was plugged with bone wax, and a larger quantity of ink introduced. The sinus was then closed. In three days, on opening the sinus again, practically the same amount of ink remained as when placed there.

Upon the application of a five per cent cocain solution to the sinus membrane, the action of the cilia was retarded for a period of about thirty-five minutes, after which time the sinus was freed of stain as before.

Next, one frontal sinus was curetted, and the other used as a control. Stain was applied to both. After two days the stain was still to be seen in the curetted sinus, though the frontonasal duct was left open; the control was free from stain. It was my purpose to ascertain the time it would require for the ciliated epithelium to regenerate, if at all, after curettment, but I was unable to keep the animal alive long enough for this to take place.

An acute frontal sinusitis was now produced by the introduction of nonhemolitic streptococci; this condition was allowed to persist for two days. The sinus was then cleansed by washing with saline solution, and again the ink stain applied. There was still ciliary action but it was sluggish and delayed. As the sinusitis subsided the ciliary action became more normal.

From my observations, I am thoroughly convinced that a healthy ciliated epithelium is a most important factor in maintaining a normal sinus condition, and our treatment should be directed toward reestablishing this healthy condition in as short a time, and with as little derangement of the cilia, as is possible.

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XLV.

SOME REFLECTIONS ON THE MAXILLARY SINUS.*

By Ross Hall Skillern, M. D.,

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THE DANGERS CONNECTED WITH NEEDLE PUNCTURE.

It is now common knowledge that a number of deaths (something under ten) have been reported and attributed to this procedure. It is also well known that the alarming symptoms in these cases always followed the preliminary injection of air. In each case, as far as we are able to ascertain, the air was forced in under heavy pressure, the untoward symptoms immediately following and death occurring in from a few seconds to twelve hours. The cause of death was attributed to an air embolus lodging in the brain. This explanation always seemed to me to lack something, to be unconvincing and far from satisfying to any one who is naturally honest in his skepticism. In the first place, when one recalls the experiments on animals, where varying quantities of air were injected into the veins which were followed by no symptoms, it is hard to reconcile this as being the real cause of death; furthermore, as postmortem evidences in the brain are practically nil, positive deductions, as far as I can learn, have never been concluded at the autopsy.

In discussing this subject let it be understood that I have never seen an actual case, so anything I might advance must be purely theoretical. Nevertheless, I have been intensely interested in the subject (being an inveterate user of the needle puncture), and have talked with many of those who have been so unfortunate as to have had one of these tragedies enacted in their presence. It would seem that all are agreed that obstruction was offered to the air at the first attempt to make an injection. To overcome this more force was applied to the piston, when suddenly the air gushed in,

*Read before the American Academy of Ophthalmology and Oto-Laryngology, October, 1925.

the patient moaned and fell over onto the floor, usually unconscious with cardiac and respiratory disturbances, the condition gradually passing away, but on the other hand not infrequently ending in death. To my mind much more than a cerebral air embolus has occurred in these cases, for in antrum puncture this can only occur where the mucosa has been forced away from the underlying bone by the point of the needle and the air can only find its way into the smaller veins, therefore no great quantity of air could have entered the vascular system from this source, hardly enough to cause an embolism. The symptoms of irregular heart, giddiness, dyspnea and cyanosis may be due to some vagus disturbance through irritation of the second branch of the trigeminal. Cocain, it must also be remembered, excites similar symptoms.

The point of the needle could not have been in the lumen of the sinus when the injection was attempted, else the air would have freely entered. Therefore, it was either buried in the swollen and inflamed mucosa or the point had pushed it away from the underlying bone and had failed to penetrate through the membrane. The sudden bursting through of the air caused a nervous shock which, in some manner, the modus operandi of which I have attempted to explain, acted upon the cardiac and even the respiratory centers with more or less serious results according to that particular case. In other words, the condition, to my mind, represents more of a shock than the symptomatology of a cerebral air embolus. Pneumatic pressure as we all know acts much more suddenly than hydraulic, and this fact could easily explain why no one has ever seen these sudden collapses following the injection of aqueous solutions. Furthermore, if only a small quantity of air became imprisoned in one of the terminal vessels in the brain, the hemoglobin in the blood would quickly absorb the oxygen in it with a rapid disappearance of all symptoms. That this is not the usual occurrence is shown by those cases which have been reported as the symptoms continued for several minutes, and in some cases as long as twelve hours before the recovery or death supervened. The great moral that this teaches is whenever resistance is offered to the air about to be injected, the attempt to force the air through

should, under no circumstances, be continued, but an investigation made at once as to the cause of the obstruction. If then despite all our efforts some impediment still remains to the passage of air, it is wise to discontinue the attempt and make a large enough opening into the antrum through the inferior meatus so that there will be sufficient room on all sides of the needle for the injected fluid to escape.

In an experience of twenty years' extensive puncturing, I have never seen a single case that gave me one moment's anxiety. This can only be attributed to the care shown in making the injection.

Another point in which all my pet theories have been revolutionized is after a needle puncture, with particular reference to the appearance of the returning fluid as a criterion of the pathologic condition of the sinus mucosa. We have been brought up as it were on the teaching that the most important and trustworthy symptom of a sinusitis was pus somewhere in the nose, and once found should be followed to its source in order to arrive at a correct diagnosis. The absence of pus, despite repeated examinations, was considered sufficient evidence that the sinus mucosa had not become infected; consequently was free from pathologic changes. This dictum applied doubly to the maxillary sinus as far as needle puncture and lavage were concerned. If pus did not appear in the washings no further thought was given to that particular sinus and the findings were considered negative, meaning that no infection was present and the sinus was free from disease. This I think was universally accepted as the proper interpretation to place upon the importance of lavage as far as its diagnostic value was concerned. Personally speaking, this tenet had become so engrafted upon the diagnosis of maxillary sinusitis that I thought it little short of actual heresy for one to even whisper the possibilities of any doubt being cast upon its infallibility.

Some years ago, however, it began to dawn upon me that despite these negative lavages, I was finding a considerable number of maxillary sinus infections which on operation showed such changes in the mucosa that could only have supervened after months, if not years, of inflammatory ac-

tivity; yet as I said, upon perhaps repeated lavage had never shown any purulent secretion. This, in my own mind, I explained away by reasoning that a spontaneous evacuation had just occurred prior to my puncture and lavage, as this had always been carried out during morning office hours and these spontaneous evacuations, when occurring, always took place shortly after the patient had arisen. These exceptions, however, became more and more frequent until my own faith became shaken, when one day, while making a lavage, a black rubber pus basin was used instead of the usual white enameled variety. I immediately noted that the returning fluid was of a light straw color, but perfectly clear and transparent, a circumstance that was not apparent when the white basin was used. Operation showed that a high grade polypoid degeneration of the mucosa had taken place.

It is said that one must cite one hundred similar cases to establish a precedent, but this one case was enough to revolutionize all my previous conceptions and overthrow the doctrines which I had persistently clung to since my first introduction into sinuology. I saw at once that the old adage, which had become almost sacred, to the effect that in the presence of a maxillary sinus infection the Lichtwitz needle could invariably be depended upon to bring out pus, was now ready for the discard. I had already seen this same thing occur with the ethmoid labyrinth when hyperplastic degeneration was finally recognized as a separate and distinct entity from the suppurative infections, and had I at that time been gifted with the slightest bit of intuition I certainly would have recognized such a possibility taking place within the maxillary antrum, which, after all, is only separated from a portion of the ethmoid by the thinnest possible osseous partition. We have all long since observed the thin watery discharge coincident with the nasal polyps (hyperplastic ethmoiditis), but who among us considered that a similar condition might exist within the antrum, giving the same symptomatology, and yet an acute observer having a repetition of a hitherto undescribed symptom (the withdrawal of a straw colored fluid) would have certainly drawn hypothetic conclusions and then endeavored to verify them by

any means at his command? Unfortunately, however, this fluid does not always appear to be available, for not infrequently the puncture is negative while at other times in the same case, as soon as the needle is introduced, the fluid begins to drop out before one even has time to adjust the connections with the syringe.

What, then, is the precise condition with which we are confronted? The exact condition is a polypoid degeneration of the mucosa of the maxillary antrum without any change in the normal appearance of the nasal mucosa even in the ethmoid region. The local symptoms will depend entirely upon the amount of hyperplastic changes which have occurred within the sinus cavity.

In the beginning and in the early part of the infection, I believe the symptoms are so light as to be adjudged a mild cold, rather chronic to be sure, or a slight vasomotor disturbance, such as one would expect from a trifling irritation.

As the hyperplastic area spreads, the symptoms, such as serous discharge, occlusion of nostril and sometimes sneezing, become more pronounced, often simulating mild attacks of hay fever. I think this will explain that hypothesis advanced some twenty years ago by the late Schadle, of St. Paul, in which he stated that hay fever was due to antral disease, and reported a number of cures by lavage of the sinus. As we see it now his cases must have been this form of antral infection.

When this hyperplasia becomes well advanced, polypi begin to show themselves in the nasal cavities and just as in hyperplastic ethmoiditis, may give rise to recurrent polyposis of middle nasal passage. Indeed Hirsch*, in a recent publication, goes so far as to state that he believes this form of disease the most frequent cause of recurrent nasal polypi.

The diagnosis in the early stages when only symptoms of a mild irritation are present can be aided by properly made roentgenograms, taken in the anterior posterior position where the two sides may be compared. The sinuologist should train himself in the interpretation of these exposures.

*Hirsch: Ueber die Entstehung der Nasenpolypen, Zeitschr. für Hals, Nasen und Ohrenheilkunde. Bd. 11, Heft 1, 1925.

In the later stages the symptoms will be so apparent that no further aid will be required if one keeps in mind the possibility of this hyperplastic condition being present.

And so in summing up, one must remember that the dangers of needle puncture are largely a personal equation with the operator himself, which by the application of proper care and patience may be avoided. Regarding the incident of simple maxillary polyposis, I am not clear, but in any event let us remember when we encounter those obscure cases of socalled vasomotor rhinitis, rose colds and the like, that we may be dealing with a case the pathology of which is a hyperplasia of the mucosa of the maxillary sinus.

XLVI.

THE RHINOLOGIC ASPECTS OF ALLERGY.*

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The rhinologic aspects of allergy may be considered under three headings, namely:

1. Nasal manifestations of general allergy or hypersensitiveness.
2. Nasal etiology of allergy.
3. Nasal treatment of allergic reactions either in the nose or elsewhere.

I. NASAL MANIFESTATIONS OF GENERAL ALLERGY OR HYPERSENSITIVENESS.

Nasal manifestations of hypersensitiveness are found in

(a) Vasomotor rhinitis (seasonal hay fever or pollenosis),

(b) Atopic coryza, perennial hay fever or chronic vasomotor rhinitis (due to inhalation or ingestion of food products or other substances, usually vegetable, or animal emanations, bacterial products, etc.),

(c) Physical allergy,

(d) Reflex reactions, which may not be primarily allergic.

In a general way the local reactions in these different classifications are the same: attacks of sneezing, frequently prolonged, accompanied by watery or mucoid discharge, sometimes almost constant and exceedingly profuse, nasal obstruction and headache. The appearances of the nasal mucosa are described by Tobey,[2] as divided into two classes. In the first there is a simple turgescence of the nasal cavernous tissue, and an increased secretion of mucus or serum, associated with sneezing. This type closely resembles acute rhinitis from infection. The mucous membrane is red. Appear-

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ances of this type are usually found in reflex disturbances, sexual, those of adolescence, etc. The second type is an aggravated form of the first but with marked pallor of all the nasal mucous membranes replacing the redness. Edema is a marked characteristic, but not an invariable or a constant one, and the pallor may disappear between acute attacks. This is the characteristic picture seen in the seasonal or perennial hay fevers. The pallor is the distinguishing characteristic that places these cases definitely in the anaphylactic group. Inspection of the nose shows great obstruction, often complete, from the edematous, pale mucous membrane. This edema does not shrink readily or, at times, at all from the application of cocain or adrenalin. The outflow of serum and mucus is constant. In the old, chronic cases, polypoid degeneration of the mucosa has occurred (in twenty-four per cent of perennial cases, according to Duke, and in about six per cent of seasonal cases).

Headache is usually caused by closing of the sinus ostia by the edema but may occasionally be the result of a general allergic reaction.

In many cases, placed by some observers as high as 25 per cent, sinus disease, either hyperplastic or suppurative, is found present in these disorders. According to Piness and Miller,[3] Todd[4] and others, these sinus conditions are merely coincidental, or directly secondary to the nasal allergic manifestations, the edema present during repeated acute attacks finally producing hyperplasia of the lining mucosa of the various sinuses by interference with the blood supply and drainage, and by closing the ostia and thereby causing them to become fertile fields for bacterial invasion and growth.

Kahn,[5] Selfridge[6] and others confirm this opinion that the sinusitis observed is caused by, but is never the cause of, allergic manifestations. The opposite viewpoint will be discussed later.

Pharyngeal manifestations of allergy are itching sensations in the throat, sometimes combined with edema of the uvula and soft palate or swelling of the tongue. The swelling may be sufficiently great to embarrass respiration. Pharyngeal cough is often present.

Aural manifestations of tinnitus and vertigo, simulating the Meniere complex, have been noted by different observers as accompanying allergic explosions, probably due to edema of the eustachian tube and tympanic mucosa.

II. THE NASAL ETIOLOGY OF ALLERGY.

2. The nasal etiology of allergy of the upper respiratory mucous membranes is represented by asthma, pollenosis, vasomotor rhinitis, reflex disturbances and similar conditions.

Great difference of opinion has existed in the past, and apparently still exists, in regard to the role played by nasal infections and anomalies in the production of the above mentioned conditions. In a previous paragraph opinions of eminent observers were given to the effect that the sinusitis, when present, stood in no etiologic relationship to the hypersensitization. Many others, however, allergists as well as rhinologists, hold the opposite view. Probably both are right to a certain extent and in certain classes of cases. Goodale[7] finds frequently in asthma and vasomotor rhinitis, particularly, of course, in those cases that do not show skin reactions to protein tests, that infection of the ethmoid and maxillary sinuses can be shown to be the etiologic factor. Such infection may be slight and not easily demonstrated, transillumination and the X-ray pictures being misleading at times, but drainage and ventilation of the infected cavities show bacterial infection and effect a cure. It seems difficult to escape the conclusion that there is a bacterial allergy in these cases, with which view both Walker[1] and Rackemann[8] are in accord. Every rhinologist of experience has done ethmoidectomies and antral drainage on asthmatic cases that have promptly cleared up and remained cured. That there have been also many failures does not, in my opinion, vitiate this idea. Many sinus operations are not successful, either immediately or even eventually, in definitely putting an end to the infection. Other sinuses may be invaded, drainage openings may close, the mucous membrane may be so badly damaged as to preclude regeneration, and there are at times outlying cells that are neither discoverable nor operable. One of the first to point out the relationship between sinus disease and sensitization as represented by hay fever was

Schadle,[9] who, in 1906, found that by lavage of the maxillary sinuses a large proportion of his pollenosis cases experienced marked relief. This does not mean that bacterial infections are the cause of allergic hay fever (all types) and asthma. No one disputes the independent hypersensitization to one or many of the almost innumerable proteins on the allergist's testing list. It does seem, however, that bacterial proteins, elaborated on the upper respiratory mucosa, must at times have a similar effect.

There is little doubt that attacks of true allergic asthma are often precipitated by an acute nasal infection. Gottlieb[10] believes that allergic asthma may be caused by accessory sinus infection and that nonallergic asthma is often due to the direct irritation caused by contact of infected secretion from the nose with the tracheal and broncheal mucosa.

Under the term atopic coryza (Coca and Cooke), Spain[11] describes nasal and eye symptoms due to a general hypersensitive state. The symptoms are identical with those of seasonal hay fever, or perennial hay fever caused by the inhalation of substances other than pollens. The diagnosis of these cases rests, as pointed out by Walker, upon the clinical history, physical examination of the nose and sinuses, and upon the cutaneous tests. Bronchial asthma is often associated with atopic coryza or perennial hay fever, due to irritation and congestion of the bronchial mucosa analogous to the condition of that of the nose and throat. Chronic infections of the nose are also frequently found associated with this condition. These are either simply coincident with the allergic condition and have only an indirect influence upon it; or, by keeping the mucosa of the upper respiratory tract in a congested or irritable state, may greatly intensify the susceptibility of the individual to those irritative substances to which, even under ordinary circumstances, he is hypersensitive. The result is a vicious circle; further congestion and irritation of all the mucosa, blocking of the ostia and increased sinus congestion. Both conditions, the allergic and the infective, need appropriate treatment.

Physical allergy is described by Duke[12] as a specific hypersensitive condition of the body to nonmaterial agents, such as light, heat and cold. It is not claimed that physical allergy

is the same as that caused by the usual material substances, but it is believed that the two conditions are closely related. Some individuals who have inherited allergic tendencies, without necessarily showing symptoms of allergy, may become specifically hypersensitive to some new substance formed in the tissues solely by the action of one of the above mentioned physical agents. Testing can be carried out by exposure to heat, cold and light, and it has been found that those sensitive to material agents are not often sensitive to physical. The symptoms produced by this type of irritant or excitant are identical with the common symptoms of allergy, vasomotor rhinitis, bronchial asthma, etc., and in the case of heat sensitization, even such slight causes as physical or mental exertion, or excitement may generate enough heat to explode the typical train of symptoms.

This is, to an extent, convincing, for we have all been accustomed, since childhood, to seeing similar manifestations, though usually mild in degree, and the old process of hardening the child to colds might have explanation on this basis. Whether these phenomena, which undoubtedly exist, can be classified as due to a true allergic condition or simply to reflex nerve action is open to question.

In the socalled "nonsensitive" (to protein substances) cases, there are two types of reaction noted by Duke,[13] namely, those in which the reaction is confined to the area exposed directly to the physical agent, and others in which reaction may involve not only the area directly exposed but distant structures as well. He cites cases where deeply breathed cold air may cause severe typical allergic asthma or vasomotor rhinitis with complete occlusion of the nose, or where a drink of cold water causes nausea, vomiting and abdominal pains.

Brubaker[14] and Sluder[15] call attention to the complex nervous system supplying the nose, containing sensory, secretomotor, vasodilator and vasoconstrictor fibres, so that the effects of a local disturbance of the nose may, by different pathways, so enormously stimulate the associated central cells as to allow an overflow of impulse into neighboring vasodilator, secretomotor and visceromotor centers and so cause respiratory, visceral and other symptoms. That asthma may be

caused by direct irritation of the sphenopalatine or Meckel's ganglion, or the fifth nerve branches, is the belief of Sluder and others, the impulses from the nose passing from the ganglion to the vidian, the carotid plexus, the lower cervical ganglia and the thoracic nerves to the lungs. Sluder[16] has precipitated asthmatic explosions by applying silver nitrate or other irritants to the mucosa covering the sphenopalatine foramen beneath which lies Meckel's ganglion, or by injecting them into the sphenoid sinus, and conversely has inhibited similar attacks by alcohol injections into the ganglion or some of the branches of the fifth nerve. Most of those that can be caused by sphenopalatine ganglion injection have their origin in an irritated condition of the ganglion caused by infections, usually chronic hyperplastic, of the posterior ethmoids or sphenoid, so that here we may have a bacterial allergy as the underlying cause of the reflex train of symptoms, or there may exist an undiscovered hypersensitiveness which makes this chain of symptoms possible. At any rate, it would seem probable, as Stein[17] suggests, that there must be some underlying sensitiveness in those individuals in which these reflex phenomena can be made manifest, for not many are subject to them. If, as believed by some writers, "polypoid ethmoiditis" is caused by the allergic condition, instead of being the cause of the allergic manifestations, it is hard to account for those cases where the asthma is relieved by the removal of polypi, the drainage and ventilation of sinuses, or even by simple cocainization of the nasal mucosa or of the nasal ganglion.

III. RHINOLOGIC TREATMENT OF ALLERGIC CONDITIONS.

Treatment of the nasal musoca during an acute allergic attack may give the patient temporary relief from distressing symptoms. Such treatment is largely confined to shrinking with cocain or adrenalin. As has already been remarked, however, the mucosa in chronic types of vasomotor rhinitis often fails to react to these agents. Cocainization of the nasal ganglion may cut short an attack of asthma if it is of the reflex type.

Operative procedures are naturally not undertaken during the acute period of hay fever or during an attack of bronchial

asthma, provided in the latter case that there are intermissions sufficiently long in which necessary surgery can be done. Great doubt has been expressed as to the value of the removal of septal spurs, correcting deviated septa, removing turbinates, or cauterizing sensitive spots on the septa or turbinates. There are times, however, when the general health of the patient may conceivably be improved by such procedures and they may, therefore, be indicated as adjunct treatment. I have seen asthma relieved immediately a number of times by middle turbinectomies. Cauterization of boggy, inferior turbinates usually gives little or no relief in chronic vasomotor rhinitis of allergic origin.

If we admit an allergic condition due to bacterial proteins, we must seriously consider the question of sinus drainage when sinus infection is associated with asthma or vasomotor rhinitis. Even if the sinus infection is secondary, common sense would indicate its elimination when possible. Goodale says that in many such cases the only way to test the influence of sinus disease on the allergic manifestations is to remove it. He further states that in many cases there is no rhinologic evidence of infection and that transillumination and X-ray examinations are negative, yet in a certain percentage of cases punctures of the maxillary antra have revealed the presence of a thick secretion. In such cases, where permanent drainage beneath the inferior turbinates has been established and the anterior ethmoid cells curetted, all symptoms cease. Sluder finds that similar results may be obtained at times, according to the nasal pathology present, by operation upon the posterior ethmoid cells and the sphenoids. That symptoms may return later, may be due to reinfection of remaining cells that have been overlooked, to the influence of acute infections on the remaining sensitized mucosa or to allergic reactions of a different type. Myer Solis-Cohen believes that all cases operated upon should, for this reason, have a course of autogenous vaccine treatment. Vaccine treatment before operation is often disappointing; after thorough removal of foci, good results are many times apparent. Vaccine made from hemolytic organisms, especially those made upon the patient's own blood after the selective method of Solis-Cohen, seem to be the most efficacious.

Whatever our opinion may be as to the causative action of bacteria in the production of allergic conditions, the fact remains, attested to by Goodale, Mackenty, Dennis, Gottlieb, Stein, Eggston and a host of others, that surgical removal of foci in the nose, and possibly in the throat, will cure a considerable proportion of these chronic or perennial cases. Their influence upon seasonal hay fever is much less apparent, though between attacks the nose should be put into as good mechanical condition as possible as a general hygienic measure. When the chronic allergic has been cured of his asthma by allergic means, not infrequently there remains a chronic bronchitis that must be combated by vaccines. In such cases it would be folly to leave an infective focus in the nose or throat to drain bacteria laden secretions into the larynx and trachea. Ordinary surgical principles indicate the ablation of such a focus.

Reflex types of asthma may be benefited by sphenopalatine ganglion injections with Sluder's alcohol phenol combination, or, if the various branches of the fifth nerve are blocked at their entrance into the nose, "there will result an inhibition of the various sensory sympathetic symptoms with a corresponding cessation of the disorder" (Stein).[17]

In no case where protein sensitization can be demonstrated should dependence be placed on nasal surgery. Where this is done, good results will rarely be obtained, and for this reason surgery for any such cases falls into disrepute. Allergic treatment should first be instituted and carried out, and then, in case of failure, surgery may be tried.

SUMMARY.

To sum up the connection of rhinology with allergy:

1. In certain types of allergy, notably vasomotor rhinitis, perennial hay fever or atopic coryza, and asthma, a nasal examination may be of great assistance in establishing a diagnosis.

2. It would seem that certain types of vasomotor rhinitis and asthma may have their origins in nasal pathology as follows:

(a) In an individual predisposed by heredity or otherwise to hypersensitization, allergic reactions in the mucous mem-

branes of the upper respiratory tract may be excited by the absorption of bacterial proteins from paranasal sinus infections.

(b) In individuals suffering from allergic reactions—asthma, hay fever and vasomotor rhinitis—due to the action of inhaled or ingested foreign proteins, such as pollens, animal emanations or foodstuffs, abnormal conditions in the nose may precipitate an attack, may increase the hypersensitiveness of the individual, or may reduce the general vitality and resistance, thereby rendering desensitization methods partially or totally ineffective.

(c) Certain forms of asthma and vasomotor rhinitis are accepted as due to reflex action from nasal pathology. These must be differentiated from true allergic reactions. Their cure lies in nasal surgery or treatment.

(d) Physical allergy—socalled—causes vasomotor symptoms from stimuli such as heat, cold and light, applied either directly to the mucous membranes or to other parts of the body. Those applied to the skin distribution of the branches of the fifth nerve are most marked.

3. Nasal treatment of allergic cases consists in:

(a) Freeing the nose from any source of infection present. This usually means draining the maxillary sinuses, the anterior ethmoid cells and at times the sphenoids.

(b) In case of asthma where sensitivity cannot be demonstrated, search for local sensitive spots and the removal of tissue that may be responsible for reflex symptoms by pressure.

(c) Proper ventilation and drainage of the nose as a general measure for restoring the patient's general health.

(d) The preparation and administration of autogenous vaccines after appropriate surgical procedures to eliminate remnants of infection and to overcome bacterial sensitization of the mucous membranes locally and the general allergic condition.

(e) Blocking the branches of the fifth nerve supplying the nasal mucosa or injecting the sphenopalatine ganglion itself.

(f) All these measures my be indicated at times to combat contributing and complicating factors, such as the chronic bronchitis of asthma and the secondary infections in the latter stages of a hay fever attack.

4. In the management of these cases of allergy, it would seem that the best results must be obtained only by close co-operation between the allergist, the internist and the rhinologist, not only in diagnosis but in the subsequent treatment.

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XLVII.

THE DIAGNOSIS AND TREATMENT OF THE ALLERGIC DISEASES.

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There are few illnesses more mysterious and elusive than those of allergic origin, both as to cause and cure. There are few conditions met with in the practice of medicine more varied as to cause, symptomatology or cure, and few which require more painstaking study if a partial or complete relief is hoped for. The diseases which are now considered of allergic origin are hay fever, asthma, vasomotor rhinitis, urticaria, eczema and serum sickness. The symptomatology complex in these diseases is extremely varied, and only after several years of observation have I been able to correlate in my own mind symptoms which may be of definite allergic origin.

I. The Orbital Symptoms.—These include puffiness and itching of the lids, redness and edema of the conjunctiva and increased activity of the lacrimal ducts.

II. The Nasal Symptoms.—Sneezing, associated with excessive watery or clear mucous secretions. This reaction may vary from slight annoyance to complete stoppage of the nose, associated with an outpouring of secretion which may drench a towel. It is often associated with frontal headache, caused by pressure in the nasal sinuses or by pain in the top of the head, occurring as a direct result of general reaction.

III. Pharyngeal and Oral Symptoms.—Itching of the soft palate and pharynx. Commonly there is edema of the uvula and soft palate and swelling of the tongue.

IV. Laryngeal Symptoms.—These include cough, hoarseness, edema of the larynx and epiglottis. This may vary in severity from simple hoarseness and cough to edema which may embarrass respiration and require immediate relief.

V. Bronchial Symptoms.—These include cough, bronchial obstruction with inspiratory and expiratory wheezing, due apparently both to edema of the membranes and spasm of the bronchial muscles, and expectoration of clear, watery or mucoid sputum, rich in eosinophils and often containing Charcot-Leyden crystals.

VI. Gastrointestinal Symptoms.—The most interesting of the abdominal symptoms is abdominal pain, which may be either mild or severe. It is often related to the eating of food and quite frequently appears a few moments after the ingestion of some particular food. The symptoms may be so severe as to cause collapse and chronic nausea, vomiting and diarrhea, which can cause emaciation in the highest degree.

VII. Cutaneous Symptoms.—There is intense itching of the skin, which may be mild and consist of simple, slight pruritis and erythema, or may be most severe, and when the skin of the face is involved, may almost obliterate the features and transform it into a swollen, red, weeping, unsightly surface. I have in my experience seen two cases, one sensitive to timothy pollen and one to ragweed pollen, both of which cases developed a definite pustular eruption, which was indeed most unsightly.

VIII. Convulsion and paralysis are rare but definite complications of allergy, mostly seen in children.

IX. Urologic Manifestations, or Irritable Bladder.—I have most frequently seen this condition existing in sensitive pollen cases, but not infrequently cases have been shown to react to certain foods. In patients having chronic bladder allergy, frequently no pathology can be found by the urologist except a hypersensitiveness to food. Not infrequently the case is misdiagnosed as cystitis or pelvic inflammatory disease.

X. Disturbances in Menstruation.—Disturbances in menstruation are rather frequently observed in patients having symptoms as a result of general reaction. I have not infrequently seen cases who have hitherto been irregular in their menstrual history become definitely regular after receiving a series of injections for pollen sensitivity, and vice versa.

XI. Hypotension.—In all of these allergic diseases it is a striking fact that the blood pressure findings are extremely

low, the average blood pressure findings in most allergic cases being from 85 to 110 systolic pressure.

XII. Eosinophilia is a fairly common condition found in the blood, although it is not as prevalent as has hitherto been taught.

In realizing that I am reading this paper before the Southern Section of the American Laryngological, Rhinological and Otological Society, I have decided to confine my remarks purely to the diseases most interesting to this society, namely, hay fever, bronchial asthma and vasomotor rhinitis. May I, therefore, present first to you the subject of bronchial asthma.

The diagnosis of the cause of bronchial asthma is not an easy task in the majority of cases, and not infrequently all the resources of the diagnostician are taxed to the limit, but if the following rules are adhered to religiously a diagnosis can be made in from 70 to 80 per cent of all cases.

I. A thorough and exhaustive history. Much can be learned from the history as to the patient's habits, his environment, his occupation, his food, and whether his attacks are seasonal or perennial in type. A thorough questioning as to the previous medical history, as many cases of asthma have been preceded by acute attacks of bronchitis, influenza, pregnancy, chronic thyroidism, and two cases have been seen following oophorectomy.

II. A complete physical examination should be made in order that cardiac dyspnea or nephritic dyspnea may be excluded. A thorough examination of the thorax should be made in order that mediastinal neoplasm or aneurysms may be excluded.

III. A complete X-ray picture of the sinuses should be made, especially if the headache is the predominating symptom.

IV. A thorough nasal and pharyngeal examination should be made if there are symptoms of nasal obstruction, or if there is constant sneezing accompanying the attack.

V. If the case is an extremely obscure one, a complete blood analysis must be done, and in several instances a high blood sugar and high blood urea have been found, denoting the beginning of organic diseases, and on instituting proper treatment a complete alleviation of asthmatic symptoms is expe-

rienced. Blood calcium estimation should be made in all cases, as a low calcium is extremely significant.

VI. Last but not least, the cutaneous test. A thorough cutaneous test, with all proteins and all pollens, should be performed. The bacterial proteins I have practically discontinued using, due to the fact that I have found them in many cases unreliable and misleading. However, occasionally I have made cultures of the separate organisms found and recovered from the sputum, and tested with these organisms with the result that positive reactions have been seen. If a case does not react to any protein or pollen after repeated tests, it has been my practice to classify that patient as a bacterial case until proven otherwise. Here let me emphasize the importance of a thorough cutaneous test. I feel sure that many cases operated upon for deflected septum, polyps, enlarged ethmoids, submucous resections, etc., could have been spared these operations and better results obtained had a thorough cutaneous test been performed. It would be a presumption for me to suggest on what types of cases operation is indicated, but I do feel that no one who is suffering from bronchial asthma should be operated upon until an exhaustive examination has been done and a thorough history has been taken. In order to support the above statement, let me cite the following statistics:

In 150 cases studied by Walker of the Peter Bent Brigham Hospital, 55 per cent were found sensitive to some protein. Of this group, horse dander was the cause in about 20 per cent, wheat protein 15 per cent, bacteria 15 per cent, pollens of spring flowers 15 per cent, pollens of autumnal flowers 10 per cent and cat hair 10 per cent. In my own experience, after reviewing a series of 100 cases, I have found that 70 per cent were found sensitive to some protein. Of this group, chicken feathers and goose feathers were the cause in about 20 per cent, eggs and milk 15 per cent, wheat 10 per cent, spring pollens 5 per cent, fall pollens 10 per cent, horse dander 2 per cent, house dust 3 per cent, tomatoes 2 per cent, dog hair 1 per cent, cat hair 1 per cent, rabbit hair 1 per cent. I have on several occasions had patients tell me that they had been tested before coming to me, and on questioning these patients I have found that they have had several proteins applied and had been told that there was no evidence of protein sensitivity.

In other words, these patients had not been thoroughly tested, due to the fact that the person doing it was not equipped with sufficient material to carry on the test thoroughly. No patient should be considered nonsensitive until every available protein or pollen has been tried. At present we are carrying about 215 different proteins. The cutaneous test is just as important in helping to diagnose the cause of bronchial asthma as any other method of precision in diagnosing any other form of disease, and when half way done is more dangerous to the patient than if it had never been done at all. I do not wish to give the impression, however, that all cases of asthma can be diagnosed alone on the cutaneous test, because there seems to exist in the minds of physicians generally a gross error concerning the value of the skin test in the specific diagnosis and conclusions which may be drawn from them. The profession at large seems to have the idea that all cases of bronchial asthma can be diagnosed alone through the cutaneous test. No idea could be more false, as only about 40 per cent of all cases of bronchial asthma can be diagnosed with this method. The method which I am using at the present time is the cutaneous method or scratch method, advocated particularly by Walker, as there is less chance of false positive reaction as compared to the intercutaneous method advocated by Cooke. The cutaneous method is carried out as follows:

A scarification an eighth of an inch long is made upon the forearm or arm, not sufficiently deep to bring the blood. The powdered protein or pollen extract is placed upon this scarification, and in the case of the powdered protein a decinormal sodium hydroxid solution is used to dissolve the protein. The appearance of a hive with pseudopods, surrounded by an irregular area of erythema, indicates a positive reaction. The reaction takes place in about thirty minutes; sometimes a delay may be experienced, and delayed reactions are just as important as the ones occurring in the allotted thirty minutes. For the sake of convenience in diagnosing, let me divide the causes of bronchial asthma into the following groups:

I. The Thermal Type.—In this type the patient complains of the asthmatic attacks occurring immediately upon leaving a warm or hot room and going into the cold, or vice versa; and again, it may be only a warm or cold draught which will pre-

cipitate an attack; or he may be walking in a cold wind, and thus bring on an attack. It has been my experience that I have seen few cases who react to nothing else but cold or heat. I have found in this type of case that the patient has been at some time in the past, or is at the present, sensitive to some pollen or animal emanation, and has developed a definite bacterial sensitivity which has become engrafted upon an already sensitive mucous membrane. I have never been able to find, through the cutaneous test, one of these cases sensitive to any food protein.

Miss L. M., aged 41 years. Chief complaint, asthma, constant sneezing and marked coryza. Twenty-five years ago patient had an attack of rose cold, which persisted until six years ago, when the hay fever symptoms disappeared. Since that time the only symptoms are constant sneezing, coryza and asthmatic attacks brought on by sudden changes in temperature, and especially on leaving the warm office and going out into the cold air outside. Also attacks may be brought on by sitting in the open trolley.

Mother has had hay fever, but no other members of the family have been affected. The patient has had several operations on the nose at different times, but has found no relief. On performing the cutaneous test upon this patient, I found that timothy reacted only slightly, but no other reaction showed. As this patient presented herself for treatment in the late fall of 1923, I decided to try an autogenous bacterin made from the nasal mucous membrane, which was given with complete relief of symptoms in six weeks from time of starting treatment.

II. The Chemical Type.—This is the type of patient who develops an asthmatic attack on inhaling any pungent odor, such as certain vapors, formalin, turpentine, ammonia, sulphur dioxid, irritating coal or wood smoke, the smoke from a gasoline motor, and even highly scented flowers and perfume. In these types of cases, as in the thermal type, I have never seen a case that did not show a sensitivity or give a history of the same to the pollens, animal emanations, and, occasionally, house dust.

Mrs. N., 36 years. Chief complaint, asthma of ten months' standing. Present illness started about ten months ago dur-

ing an attack of grippe and severe bronchitis. The attacks are intermittent, occurring only when she feels chilly, or as if an attack of grippe were coming on. She has been in the habit of taking a grippe capsule, which had been prescribed for her, and immediately the attack of asthma would appear. The prescription contained aspirin, and upon instruction never to take aspirin again, the patient has been free of asthma.

III. The Neurotic and Atmospheric Type.—In this type the patient is highly neurotic, and asthmatic attacks are precipitated by fright, sorrow, hysteria, or any sudden emotional outburst; and in the atmospheric type is seen the patient who develops an attack of asthma just before a thunderstorm or any sudden change in atmospheric pressure. These two types are extremely difficult to diagnose, but in my experience these patients have in several instances shown a weak sensitivity to pollen or animal emanations, plus a markedly depleted physical condition, due to some long standing infection or general mental strain. I believe these cases can be classed among the cases of endocrin dysfunction.

IV. The Pollen Type.—There is little to say concerning this type, as the cause is easily found by the cutaneous test. This type may be perennial as well as seasonal. Do not overlook the fact that there is, in almost all of these cases, a secondary bacterial infection of the mucous membrane, and I believe that in almost every instance the cause of the asthmatic attacks in the pollen type is due to bacterial invasion, plus the presence of the pollen itself. I feel that the constant bombardment of the mucous membrane by the pollen lowers the physical resistance of the patient, and the mucous membrane is made a fair field on which the bacteria may grow in profusion, with a consequent bacterial protein sensitivity produced. In order to carry out my theory, I should add that asthmatic attacks are rarely seen occurring until at least three or four weeks have elapsed after the hay fever begins.

V. The Animal, Epidermal and Food Protein Type.—In this type are classified the patients sensitive to the food and animal proteins. If the asthmatic attack is caused by the animal proteins, it will readily be shown by the cutaneous test. On the other hand, if it be food protein sensitivity the offending pro-

tein is not always easy to locate, and, as I have said before, an exhaustive study should be made before the patient is considered nonsensitive. It must be borne in mind that the absolute withdrawal of food from the diet may give a negative test. This should be remembered, as the evidence that a particular food is causing the trouble may be so great that the food will be withdrawn, and a later test may give a negative reaction to that food.

VI. The Bacterial Type.—In this type of patient the cutaneous test is negative throughout, although there may be a history of hay fever or asthmatic attacks being caused by contact with animals. Almost always there is a history of asthma. The method of diagnosis in these cases is one of elimination alone, and no case should be diagnosed a bacterial case until all methods of diagnosis have been exhausted. There is no doubt in my mind, however, that a bacterial sensitivity may be engrafted upon, or may be the direct cause of, asthma in any of the foregoing types classified above.

TREATMENT.

Let us now consider the treatment of these different types of asthma.

The treatment of the chemical and thermal type is practically the same. If the cutaneous test shows a positive reaction to any of the proteins or pollens, a thorough desensitization with the pollens causing reaction, plus an autogenous vaccine, is required. If animal protein causes reaction, resort must be had to desensitization with that animal protein causing protein reaction; if food protein, the protein causing reaction is to be removed from the diet, also potassium iodid in 10 grain doses after each meal is given to increase bronchial secretion and facilitate freer expectoration, and also to break down the barriers surrounding the foci of infection in the lung tissue, thereby increasing the activity of the action of the vaccine. General supportive treatment must not be overlooked.

The Treatment of the Neurotic or Atmospheric Type.—In this type the condition is almost always based upon a sensitivity to some form of protein, which may be demonstrated by the minutest reaction, but in these cases are found the types which are highly neurotic and emotional and show irregularity of

menstrual history. These cases, I believe, are definitely of endocrin origin. They may or may not show sensitivity to reaction, but it is my procedure in these cases immediately to try to build up their physical condition, to better their mode of living, to order a good, supportive, nourishing diet, moderate exercise, one or two quarts of milk a day, and no competitive exercise. If there is a sensitivity to any protein, desensitize; or if food, remove that food from the diet. Again, a sputum is collected, and an autogenous bacterin is made and given. I have found in these cases that a combination of suprarenal extract, 2 to 5 grains, and postpituitary extract, grains ¼ to ½, three times a day after meals, and if hypothyroidism is suspected, thyroid extract in ½ to 2 grains, in conjunction with the above prescription, gives the desired relief.

The Treatment of the Pollen Type.—In the pollen type of asthma the treatment is comparatively easy, as in the majority of cases the hay fever symptoms are also present and the treatment is the full course of the offending pollens. In the average case, these injections will take care of the asthmatic attacks. If, however, the history shows that in previous years the asthmatic attacks have been extremely severe, and have continued long after the hay fever symptoms have subsided or disappeared, or if the only remaining symptom is a cough or constant sneezing, an autogenous bacterin is made from the sputum and from the nasal mucous membrane, and the bacterin is used in conjunction with the pollen extract injections with most satisfactory results.

Last year I saw one case in which the only symptoms were those of asthma. This asthma occurred, and had occurred, for several years, beginning about August 20 and lasting through the fall hay fever season. On testing this patient for the fall hay fever pollens, I was surprised to find that she did not react to anything. I therefore took for granted that she had sufficient resistance developed to show no sensitiveness to the cutaneous test, so I gave her a full course of ragweed injections, with the result that she went through the season without one attack of asthma.

Often these pollen cases will be left with a definite sensitiveness of the nasal mucous membrane after the season. In these cases it has been my practice to have an autogenous vaccine

made from the nasal secretions and a course of injections given, with the result that the condition is soon alleviated. Nasal obstruction and spurs have quite frequently been found in these types of cases, and it is needless to say should be removed if diagnosed.

The Treatment of the Animal, Epidermal or Food Protein Type.—The treatment in these types of cases is different and should be taken up separately. The animal epidermal cases will be easily diagnosed by the cutaneous test. The removal of feather pillows, quilts, etc., from the beds will give miraculous results. If unable to remove the cause or the offending protein, a course of desensitization with the protein causing reaction will, in the majority of cases, give the desired result. In the case of the foods, I have been unable to desensitize any patient against the offending protein, due to the difficulty in getting the patient to cooperate in the treatment, and the offending food is dropped from the diet for a year, with the result that many have developed immunity.

Treatment of the Bacterial Type.—If after all methods of diagnosis have failed to show the causes of the asthmatic seizures, then that case is classified as a bacterial protein sensitivity case, and these cases are treated with an autogenous vaccine. The sputum is collected and a nasal smear is taken and a polyvalent vaccine is made. This should be done under the most antiseptic conditions possible. The Cohen-Heist method has been used in the majority of our cases in preparing the vaccine, with the result that I believe that our results have been slightly higher in their percentage of cures. Injections of the vaccine are given in increasing doses at five day intervals. If definite relief is not experienced within six to eight weeks of starting the treatment, the patient is retested for possible sensitivity which might have been missed, and a reculture of the nasal secretions and sputum is made. Potassium iodid is again given in grains 10, three times a day, after meals, for the same reasons as stated in a previous paragraph of this paper. Again, the patient must be impressed with the fact that no competitive exercise, or exercise which is associated with fatigue, should be indulged in.

Mention should be made here of the importance of a blood calcium estimation being made in all asthmatic and hay fever

patients. Brown and Hunter have shown in a series of 166 consecutive patients on whom calcium determinations were made 39 per cent had a definite calcium deficiency, 24 per cent were low normal, and 37 per cent were normal. There were 100 patients with asthma, of these 42 per cent had a definite calcium deficiency, 23 per cent were low normal and 35 per cent were normal. There were 43 patients with perennial hay fever or vasomotor rhinitis. Of these, 37 per cent had a definite calcium deficiency, 21 per cent were low normal and 42 per cent were normal. There were 32 cases of typical seasonal, or pollen, hay fever. Of these, 28 had a definite calcium deficiency, 34 per cent were low normal and 38 per cent were normal. There were 15 cases of eczema. Forty per cent of these were calcium deficient, 20 per cent were low normal and 40 per cent were normal.

All patients with any degree of calcium deficiency were advised to eat plenty of the foods that are rich in lime, and to get as much sunshine as possible. The foods that are particularly rich in calcium are eggs (yolk chiefly), fresh milk, cheese, whole wheat or bran bread, oranges and other fresh fruits, carrots, cabbage and other green leafy vegetables, oatmeal, dried beans, prunes, almonds, walnuts, peanuts. Patients with a low normal calcium were all given calcium lactate from 3 to 10 grains, three to four times a day, and parathyroid, 1/20 to 1/10 grain, from two to four times daily, or thyroid, from 1/10 to ½ grain, from one to three times a day, or both, by mouth. Patients with a definite calcium deficiency also received the quartz lamp treatment. Gratifying results were obtained in 54 per cent of the calcium deficient cases; definite improvement in an additional 43 per cent, and in only 3 per cent of the cases was the treatment a failure.

I have not had to date the vast experience of the above mentioned writers, but the small number of patients under treatment and who have been treated with the quartz lamp and calcium lactate have shown decided beneficial results with this method.

TECHNIC FOR MAKING BACTERINS.

Sputum.—Carefully select from a fresh sputal mass some thick mucopurulent material and wash thoroughly in three

or four changes of sterile physiologic salt solution in Petri dishes. After last washing transfer a portion the size of a wheat grain to a hemoglobin agar plate and spread in sections so as to have increasing dilutions on the plate. That is, gently spread the sputum with the aid of a platinum spatula over about one-fifth of the plate surface, stroking over and over again until the mass is well and equally spread. Sterilize the spatula. Spread a small portion of this fifth over another fifth, using the same care to get an equal distribution. Sterilize the spatula. Repeat from the second fifth to the third fifth, and so on, thus making five dilutions on the plate.

This is done because different specimens contain varying numbers of viable organisms, and it is necessary that some part of the plate shall have few enough organisms present that their cultural characteristics shall be easily differentiated.

Make two smears of the washings. Stain one for tubercle bacillus, the other by Gram's method and note findings.

Incubate 36 to 48 hours, or long enough for complete differentiation of types.

Pick colonies according to their characteristics and transplant to hemoglobin agar slants, taking one of every type present.

Incubate twenty-four hours.

Make smears of various types picked and stain by Gram's method. Note characteristics. At this time many types can be named. That is, by combining the information obtained from their morphologic and cultural characteristics a pneumococcus or micrococcus catarrhalis could be recognized, but for other types like the streptococcus group, Friedlander bacillus, etc., it is necessary to investigate their action on other media, such as the sugars, etc., for final naming.

Make two or three hemoglobin agar slants of each type isolated, using two for the free growing types and three for the less free growing types.

Incubate twenty-four hours.

Add about 1 cc. of sterile physiologic salt solution to each type, and with the aid of a platinum loop rub off the growth from the surface of the media, taking care not to break the surface.

Transfer the suspensions thus made into sterile dry tubes, using a separate tube for each type.

Heat in water bath to 60 C. for one hour on two successive days.

Count the number of organisms present in each type and combine them to form the dosage desired, and place mixed suspension in sterile 15 cc. bottle and stopper, with some type of rubber stopper that can be punctured by a hypodermic needle. Shake the bottle in a mechanical shaker for three to six hours.

Test for sterility by withdrawing some of the contents of the bottle by the aid of a hypodermic needle and syringe and planting on hemoglobin agar slant.

Incubate forty-eight hours, and if at that time there is no growth on the slant remove stopper aseptically, add one drop of tricresol and restopper. It is ready for use.

Postnasal and Tonsil Cultures, etc.—With the aid of a small sterile swab collect some postnasal secretion or secretion from a tonsillar crypt, taking care not to brush the anterior nares or any part of the mouth or throat any more than is necessary.

Immediately transfer to a hemoglobin agar plate, spreading in increasing dilutions as described for sputum.

Make smears and stain by Gram's method, noting the findings. Continue as in sputum.

Although many types will grow well on plain agar, the virulence, if any, of the types is better supported by keeping them on hemoglobin agar at all times during the process.

Use all types present, whether recognized pathogens or not, because nonpathogenic types may under certain circumstances be pathogenic. In any event, we are at least adding a nonspecific foreign protein that will probably aid the specific types.

The collection of the specimen and the selection of a protein of the specimen for culture is of the greatest importance, and on this depends to a great degree the value of the resulting bacterin as a therapeutic agent.

HAY FEVER.

Since 1565 the condition known as rose cold has been recognized by the profession practicing the healing art, but the medical world is indebted to Dunbar[1] for the exhaustive

scientific proof, in 1903, of the specific action of pollen as a cause of hay fever. Dunbar's work opened the way to a new method of treatment founded upon immunology. John Bostock,[2] in 1819, however, described the symptom complex of hay fever, but it was not until 1873 that Blackley[3] determined the real cause of the symptoms, namely, the pollen of plants. Curtis,[4] in 1900, was the first to attempt to produce active immunity or to treat the condition; he used extracts of the whole plant.

It would be impossible to mention the names of all those who have been writing and reporting on the condition in the past ten years, but the mention of the names of I. Chandler Walker, Freeman, Koessler, Cooke, Goodale and Selfridge is justified, since it is these men who have done most toward establishing the treatment of seasonal hay fever.

Dunbar isolated from the pollen what he thought was a true toxin, but later investigation established the fact that the active substances of the pollens are proteins with active antigenic properties. It is now possible to determine the specific pollen causing the hay fever symptoms in each case, and the condition can be alleviated, and in many cases prevented by treatment with an active antigenic extract prepared from the causative pollen.

It has been shown by the American Hay Fever Prevention Association that about 1 per cent of the population of the United States is susceptible to hay fever (about 1,100,000). The disease is common in the United States and in England, but is comparatively rare on the continent of Europe. It occurs only in regions in which flourish plants rich in pollen, and only when those plants have reached the flowering stage.

Hay fever is a condition of hypersensitiveness to pollen proteins, and is produced primarily by the inhalation of wind borne pollens. Only those wind borne pollens that are inhaled during normal respiration cause hay fever.

Scheppegrell[5] says: "While there are many plants whose pollens may cause hay fever reaction when applied to the nostrils, only pollens which float in the air and can reach the nostrils in the course of normal respiration are responsible for true hay fever. This fact was definitely established by means of atmospheric pollen plates, which were exposed daily, during

the hay fever season, at various stations. The plates were coated with glycerin and exposed for periods of 24 hours and then taken to the laboratory, stained, examined microscopically and the pollens identified.

Although wind borne pollen is so widely distributed that the mucous membrane of every person comes in contact with it, yet only those sensitized develop hay fever. Just what causes this sensitiveness is not known, but I believe that it is largely due to a definite hypoadrenia, as all hay fever subjects show a definite symptomatology of lowered suprarenal function. However, this sensitiveness to pollen protein is greatly increased by the amount of the pollen inhaled, by nasopharyngeal disease or obstruction, by the lowering of a person's resistance, and also there seems to exist a definite hereditary sensitiveness to hay fever in certain families.

Ulrich[5] has pointed out that the sensitive membrane may have lost the faculty of rapidly converting the proteins into aminoacids or may never have had it.

It can be proven positively by the cutaneous test with pollen extracts that each hypersensitive patient is definitely sensitive to a particular pollen.

Pollens are carried from one plant to another through the agency of the wind or by insects. The principal and most important factor in connection with hay fever is that only those pollens which are carried by the wind and which are therefore in the air can cause true hay fever.

Wind pollinated plants are distinguished by their inconspicuous flowers, which are devoid of scent or odor; their pollen is shed in large amounts and is light and buoyant.

On the other hand, insect pollinated plants are characterized by their bright colored, conspicuous flowers, which have a sweet scent or odor; their pollen is heavy and viscid, and is shed very sparingly.

There are certain insect pollinated plants whose pollen will undoubtedly produce hay fever symptoms, if directly inhaled, but such pollens are never found in the air in any great quantity, and so are practically not a factor in the hay fever situation. The pollen of insect pollinated plants may precipitate an attack of hay fever under very exceptional circumstances; such, for example, as when the flowers are deliberately smelled,

as might be done, particularly by children, or where the plants are grown in great profusion, in close contact with the patient, or even, in some cases, where the flowers are used for room decoration, but such symptoms would not long persist. The fact that true hay fever is in the large per cent of cases caused by the inhalation of the wind borne pollens only, eliminates the long list of insect pollinated plants which are popularly supposed to cause hay fever. The insect pollinated plants comprise alfalfa, clover, roses, dahlia, honeysuckle, rose of sharon, chrysanthemum, lily of the valley, aster, clematis, lilac, goldenrod, dandelion, white daisy, yellow daisy, sunflower, mountain laurel (rose bay or oleander), sugar beet, fruit trees (apple, apricot, pear, peach, plum) and several other trees, including alder, fir, hemlock, pine, spruce, tamarisk (or flowering cypress) and willow.

Hay fever in the late spring and early summer is often erroneously designated rose cold or rose fever. Such a name is a misnomer, for hay fever at this time of the year is not caused by rose pollen, but by pollen of the grasses. In like manner, fall hay fever is popularly attributed to pollen of the goldenrod, while in reality it is caused, in the majority of cases, by the ragweed, dwarf and giant, and eupatorium perfoliatum.

The pollen of corn, in spite of its toxicity, is rarely responsible for hay fever, because its size is so great that it cannot travel far; so, only by close proximity to a corn field can hay fever be induced. The above holds true for rye, oats and wheat.

Causes and Seasons of Hay Fever.—The hay fever seasons vary in different sections of the country, depending upon the flowering periods of the various hay fever plants, which are governed by climatic conditions.

In the following tables the most important pollens from a hay fever standpoint are indicated by an asterisk (*); the others are secondary, or of minor importance.

Diagnosis.—The diagnosis of hay fever or rose cold is not a difficult problem, as the symptoms are rather classical: conjunctivitis, coryza; itching throat and mouth and nasal mucosa; cough; some fever in the more severe cases; gen-

eral malaise; and not infrequently asthmatic attacks coming on in the latter part of the season, and sometimes as early as the beginning of the second week. In a small percentage of cases the only symptoms may be a persistent cough, or definite asthmatic seizures coming on at certain periods of the year. I have not infrequently seen cases of pollen sensitization overlooked because the classical symptoms were not present.

Cases of spring hay fever have been observed as early as April 15, and are usually due to the pollen of the maple, willow, walnut, ash, and the fruit trees; but May 15 is the usual date when spring hay fever begins and is the date when June grass pollinates. The next in order of pollination are the dandelion, orchard grass, red top, timothy, daisy, rye and oats, which carries the trouble into July. Corn and other cereals must be considered.

The fall hay fever begins on or about August 15, and is usually caused by the pollens of the dwarf and giant ragweed in the majority of cases; goldenrod in about 5 per cent of cases; sunflower in 2 per cent; goldenglow, cocklebur—and last but not least, a pollen which I isolated as causing marked reaction in about 10 per cent of cases—the eupatorium perfoliatum. This plant or weed is of the ragweed family, and pollinates about September 15, and is, I believe, greatly responsible for the aggravated symptoms and asthma coming on the later weeks of the hay fever season. This was proven in 20 cases who were sensitive to eupatorium and had definite seizures the season previous. They were treated with eupatorium extract, with the result that none had any asthma and 4 cases only slight hay fever symptoms throughout the season.

Before he can rationally treat hay fever, the physician should familiarize himself with those pollens which are prevalent in the vicinity where he lives, so that he may intelligently select the proper pollens to use for the test, for patients may exhibit skin reactions to pollens to which they are not or never will be exposed.

After the history has been obtained and a conclusion reached as to the possible offending pollen, the cutaneous test is then resorted to. The test which is most extensively used, and

which I feel is most reliable, is the cutaneous test, and is performed in the following manner: The flexor surface of the forearm is cleansed with alcohol or ether, and a number of small cuts are made upon this surface. These cuts should not be deep enough to draw the blood, or the pollen diagnostic material will be washed away from the scarified tissue. A small amount of the pollen protein is then placed upon these scarifications. After the lapse of twenty or thirty minutes the material is washed off, and there is a positive reaction, a raised white elevation or urticarial wheal surrounding the cut will appear.

Rarely one will test a case who reacts to all the pollens applied. In such a case there is a marked irritability of the skin, and such reactions should be ignored, and the test performed at a later date, when a different reading will be found. It is not infrequent, however, to find a patient reacting to more than one reagent: it is, in fact, rather unique to find a patient who reacts to only one pollen.

A positive reaction indicates that the person is sensitized to the protein of the pollens causing reaction, and is, therefore, susceptible to hay fever if he comes in contact with that species of pollen. Several skin tests may be made with the principal pollens to which the person may be exposed and treatment given with the pollen extract corresponding to that pollen which gives a positive reaction to the test.

Too much cannot be said about the care which should be exercised in making the small scarifications upon the forearm. By that I mean that they should be uniform in size, each one about one-eighth of an inch in length. If the scarifications should not be uniform in size, one long and one short, for instance, the long scarification will give a much larger reacting surface, and therefore a larger reaction, and hence a false reading will be obtained.

The method which I use is to take a sharp pointed scalpel, and turning it sidewise use the side of the point and not the cutting surface; then with a steady downward draw of the point the upper layer of the skin is torn and the under surface laid bare. If the cutting edge is used it will be difficult not to draw blood; this, as I have said before, must be avoided. After all the necessary scarifications have been

made, the extract to be used is dropped upon the cut with the aid of a flat ended toothpick held in a perpendicular position after being withdrawn from the bottle. Fifteen to twenty minutes are then allowed to elapse before a reading is made.

TREATMENT.

Clowes was the first to report a definite method of vaccination against hay fever in this country; but to Koessler must be given the credit for placing the method of desensitization against hay fever on a scientific basis, for he was the first to report detailed methods of preparing pollen extracts.

It has been noticed, after careful observation, that the timothy and ragweed pollens are the most resistant to treatment, the reason being that timothy and ragweed are the most luxuriant growths, and also, their pollens are the lightest and, therefore, most easily blown about by the wind.

The method of making the pollen extracts is as follows: To 0.5 gm. of the dry pollen is added 44 cc. of sterile physiologic sodium chlorid solution, and the mixture is shaken thoroughly at frequent intervals for 24 hours, after which enough absolute alcohol (6 cc.) is added to the mixture to make the alcoholic content 12 per cent. Again the mixture is thoroughly shaken at frequent intervals for 24 hours, after which it is centrifuged at high speed and the supernatant fluid is pipetted off and saved. This supernatant fluid, therefore, consists of the pollen protein dissolved in a 12 per cent alcoholic physiologic sodium chlorid solution, and it represents, by weight, one part pollen to 100 parts solvent. This 1:100 solution is used as stock, and from it other dilutions—1:500, 1:1,000, 1:5,000 and 1:100,000—are made, using a 12 per cent alcoholic physiologic sodium chlorid solution as diluent. These solutions are used not only for skin tests, but also for treatment, and with the addition of a small crystal of thymol they will keep for months in a cool place.

TYPICAL CASES—PROPHYLACTIC OR PRESEASONAL TREATMENT.

The importance of preseasonal pollen treatment cannot be too strongly emphasized. For successful desensitization, it is essential that the prophylactic treatment with the pollen extract be started about three months before the date of the

expected attack of hay fever. It has been my procedure to test all cases of hay fever, whether they be spring or fall types, during the last two weeks of February. My reason for testing all cases at that time is that some cases which have prior to that time shown symptoms of a spring or fall type alone may then show a definite sensitivity to other pollens, which sensitivity may have developed in the past year. In such a case, desensitization should be carried out for all the pollens reacting.

Having determined by the diagnostic test the pollens to which the person is sensitive, the reading is made and the pollens are marked upon a chart which corresponds to the location of the tests upon the forearm.

Let us, for example, consider the patient who reacts as follows: June grass, 3 plus; red top, 2 plus; orchard grass, 2 plus; timothy, 4 plus. My course of treatment would be to combine all four pollens and use the extract for desensitizing. If, however, the reactions were as follows: June grass, 4 plus, and timothy 4 plus, with the history that the patient had hay fever symptoms starting on or about May 15 and lasting a week or two with a week or two of remission of symptoms, and again starting about the latter part of June or the first of July, and then the symptoms recurred and lasted through July; I would treat that patient only with June grass and timothy.

Injection of the spring pollen extracts will not desensitize against any of the fall types. They must be treated separately.

In a small percentage of persons (about 10 per cent) the complete prophylactic treatment with the pollen extract will fail to establish complete desensitization, and such a person will not be fully protected from hay fever symptoms. Under these circumstances the hay fever attack may be delayed beyond its usual date of onset; or the attack may occur at its regular time, but the symptoms will be mild and will not last long. For such persons it is my procedure to continue increasing the injections gradually, until there is a definite alleviation of the symptoms. I have seen cases completely relieved after one or two additional injections.

A few persons (less than one per cent of my cases) may be unusually sensitive to pollen protein and the initial dose for the average person cannot be administered for fear of reaction. Here the initial dilution must be much weaker, necessitating, of course, additional doses, and a greatly prolonged time of treatment.

TREATMENT OF THE ATTACK.

Preseasonal treatment yields by far the most satisfactory results, and the advisability of taking the pollen treatment in advance of the hay fever season should be thoroughly impressed upon the patient. Nevertheless, many patients do not apply to the physician for relief until the hay fever symptoms are well advanced. Treatment with the pollen extract during the pollinating season has proven beneficial in many cases. In treating this type of case, it must be constantly borne in mind that the patient is already absorbing pollen from the atmosphere, and in consequence great care should be taken to start with the weaker dilutions, and increase the dosage with caution. The interval of dosage, however, can be, and should be, shortened in order that the required amount can be given in the limited time at the disposal of the physician. It may be necessary to administer the full course of treatment before the symptoms entirely disappear in some patients; others may be benefited by three or four injections, with complete cessation of hay fever after the eighth or ninth dose. The doses may be given at intervals of three or four days instead of a week. When used during the attack of hay fever, pollen extract has in many cases alleviated the condition, as indicated by the symptoms being milder and the attack shorter.

The physician can do much to advance the science of the pollen treatment in hay fever if he will use the opportunity afforded him when administering pollen extract during an attack, to impress upon his patient the possibility of greater benefit to be derived from preseasonal treatment in the following year.

There are at present two methods of treatment: the short interval method and the longer interval method.

The short interval method as advocated by Duke, Kansas City, is as follows: The injections are given twice daily in the beginning of treatment, and the dose doubled at each inoculation until the stronger dilutions are reached. At this time, it is frequently necessary to span out the time interval to daily injections or to injections given at two or even three day intervals. The treatment is started for the fall cases about July 15 and carried through to September 15, when treatment is discontinued.

The longer interval method: Here the treatment is begun at least two or three months before the season begins, and ends with the beginning of pollination, and the injections are given over five day intervals.

The latter method I am practicing and in my experience I have had much better results with this method.

DOSAGE AND ADMINISTRATION.

In the application of desensitization to hay fever, it is important to always begin with the weaker dilutions and smaller doses.

The experiments of Weil[7] have proved that small amounts of protein injected repeatedly will desensitize against small amounts, whereas the injection of large amounts of protein will desensitize against large amounts. Hence, the method of gradually increasing the dose is theoretically correct, and the person must be rendered refractory to large doses about the time of the expected attack of hay fever. In its practical application, also, this method has been proved to be correct.

Let us now consider the actual dosage and treatment of a case, be it spring or fall type. Remember that the spring hay fever usually starts on or about May 15 and is due to the grasses; the fall type begins on or about the 15th of August, and is due usually to ragweed, short and long. However, goldenrod, goldenglow, cocklebur, sage brush, etc., may be causative factors.

After the readings of the cutaneous tests have been made, and the pollen or pollens for treatment decided upon, the extract of the pollen or pollens causing reactions is made up in four dilutions of varying strengths, i. e., 1:10,000;

1:5,000; 1:1,000 and 1:500. Again the cutaneous test is resorted to in order that the initial dose can be ascertained. Four scarifications are made, with a control, upon the forearm and a drop of each dilution is placed upon the scarifications. The cut showing a reaction to the weakest dilution shows the dilution which should be used for the initial injection, or, using a still safer method, the strongest dilution giving no reaction. This latter is my usual procedure. Now that the dilution to be used is ascertained, the first injection given is two minims, and every five to seven days an increase of two minims, until six minims of that dilution have been given; then the same increase in dosage with the other dilutions until six minims of the 1:500 is reached. Then it is my procedure to continue giving the 1:500 dilution in ascending doses of one minim each, until a dose of 15 minims is given. If the case shows signs of hay fever after the season starts, I continue with this 15 minim dose.

If a general reaction is experienced, eight or ten minims of a 1:1,000 adrenalin chlorid solution should be injected into the arm as quickly as possible, and almost instantaneous relief will be experienced by the patient. A patient should never be allowed to leave the office for at least twenty minutes after the injection, for fear of a reaction.

In cases where the results have not been 100 per cent cure, and conjunctival symptoms are present, a drop of castor oil or "estivin" in each eye has been most pleasing to the patient. Where there is a slight coryza, a combination of the following is most beneficial: camphor and menthol, a. a. gr. 5; adrenalin inhalant, one dram; ol. olivae q. s. ad one ounce using gtts. 5 in each nostril four or five times daily or when going out of doors.

ATYPICAL CASES.

Let us consider the socalled perennial type of hay fever, the case having typical symptoms of hay fever at any time of the year. There is, in almost all such cases, a definite history of seasonal hay fever attacks, which, as time goes on, finally develop into the type mentioned above. These attacks may be precipitated by thermal, chemical, protein (animal or food), or pollen irritation, or by house dust. It is

my procedure, in these cases, to first get a complete history, to follow that by a complete test, and last but not least, to have a thorough nasal and laryngeal examination of the patient to ascertain if there be any nasal obstruction. If there is marked nasal obstruction it should be removed, but bear in mind that too hasty operative measures should not be resorted to, as many an already sensitive nasal mucous membrane has been made hypersensitive by a too zealous operator.

The next step is to obtain a culture from the nasal mucous membrane and the sputum, and have an autogenous bacterin made. If the patient has shown any reaction to the pollen extracts, the extract of that pollen and the bacterin combined are given hypodermically in ascending doses. If, on the other hand, the patient reacts to some of the animal proteins, such as chicken feathers, goose feathers, dog hair, cat hair, rabbit hair, horse dander, sheep wool, etc., these should be eliminated from the patient's surroundings. At the same time, a good supportive tonic and a capsule containing suprarenal extract, pituitary extract and thyroid extract, gr. 2 of each, should be given three times a day. I believe that there is a general depletion of the resistance of the mucous membranes of the respiratory tract.

Another atypical type of pollen sensitivity is that in which the only symptom may be asthmatic attacks. Here a complete history of the case will be of great help, and the asthmatic attacks will be shown to be seasonal in character. Again the cutaneous test should be resorted to and if a positive reaction is obtained, the full course of injections of the reacting pollen will give most beneficial results.

Another atypical type of pollen sensitivity is seen in the case where there are no hay fever symptoms, but where there is a marked sensitivity of the skin, with the production of urticaria or erythema, even becoming pustular. This type is extremely rare; I have seen only two such cases in my experience, and have never seen a description of any such phenomenon in the literature. There was, in both cases, a definite history of poisoning of the skin after coming in contact with ragweed, in one case, and the spring grasses in the other. After the test was found positive, a full course

of treatment was given, with complete alleviation of symptoms.

As a final word to those who may be contemplating taking up this work, let me emphasize the following points:

1. Be sure to obtain a complete history. It will aid in deciding what pollen or pollens should be used in treatment.

2. Be careful of technic in making the cutaneous test. Contamination may be most misleading, and the desired results will not be gained. In making the scarifications, be sure that they are of equal size, and not too close together. Misreadings may result from the former, and if too close together the reactions may unite, making it impossible to note which is the major reaction.

3. Never allow a patient to leave the office after an injection of pollen extract has been given, for at least fifteen minutes, for fear of a reaction.

4. Never give an injection of pollen extract without a 1:1,000 adrenalin chlorid solution close at hand. If a reaction occurs, give immediately five to ten minims hypodermically.

5. Constant encouragement must be given the patient throughout the course of treatment, as he is usually pessimistic. It will have a marked influence on the end result.

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XLVIII.

THE NATURE OF HAY FEVER AND BRONCHIAL ASTHMA.*

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No one today seriously questions the statement that hay fever is caused by pollens, and so many plants and trees have been found of etiologic importance during the past ten years, as a result of intensive studies in this disease, that it is safe to say that any plant which sheds pollen can cause the disease. In the minds of many of the laity hay fever is still believed to be caused only by roses, but it is now known that this is but one plant of a very large number that may be responsible. On the other hand, the pollens of so many grasses are implicated in the spring and early summer types that the designation of "hay fever" is well chosen, although the term "pollen disease" would appear to be more suitable.

Every year the list of pollens is increased as experience with them is gained in relation to the etiology of hay fever. This is not strange when one remembers that the floras vary in different parts of the country, and those physicians interested in the diagnosis and specific treatment of the disease should be particularly acquainted with the etiologic plants of their vicinity. As I shall discuss shortly, it is not at all uncommon to observe hay fever victims yielding positive skin reactions to the pollens of plants which do not grow in the vicinity of their homes and spheres of activity, and in instances of this

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kind it is hardly required that their extracts be used in specific treatment by desensitization. Much confusion also results from the fact that several names are sometimes given the same plant or several totally unrelated plants share the one English name. As an example of the former, one may mention that very common offender, "ragweed," designated in some localities as horseweed, pigweed or bitterweed. As a general rule, however, the pollens most frequently producing hay fever in the United States may be grouped under those of the grass, goosefoot, pigweed, ragweed and thistle families.

In order to produce hay fever the pollens must come in contact with the mucous membranes of the eyes, nose and respiratory tract, and the method of their transfer from the anther of one plant to the pistil of the same or of a neighboring one is of some importance in relation to hay fever. For example, plants like the grasses and ragweeds, which depend upon air currents or winds for the dissemination of their pollens, are the greatest menace to hay fever patients, because of the wide dissemination of the pollens, and these are the most important etiologic agents in hay fever. In this connection, however, the question of size and weight of the pollens is of importance since the lighter pollens are more widely distributed than the heavier ones, which thereby explains hay fever attacks occurring when the victim is a long distance from the plant. Insect pollinated plants, like the rose, daisy and dahlia, come next in importance, because their pollens may become atmospheric in some degree, but among those plants, like most varieties of cultivated wheat, which are self pollinated, the pollens are of least importance in relation to hay fever because of their very limited distribution.

As is well known, attacks of bronchial asthma may occur in the course of hay fever due to the access of pollens to the bronchi. But this is only one and the least important cause of bronchial asthma, since it is now quite well established that this disease may be caused by a very wide variety of protein agents, including not only those of the foods, but, more importantly, the epidermal proteins of the lower animals, including both mammals and fowl, in addition to those of furs and such additional substances of animal origin as glue and silk, as well as such substances of vegetable origin as tobacco, orris root,

henna, hops, cotton, the bacteria, etc. Indeed the list of etiologic agents in bronchial asthma is far larger than in hay fever, and for this reason the exact etiologic diagnosis and specific treatment of the disease is fast and properly becoming a specialty in medicine because of the large number of skin tests frequently required before the etiology of some cases is successfully revealed.

But while clinical experience has firmly established these facts in relation to the etiology of hay fever and bronchial asthma, as well as suggesting that a similar mechanism is apparently sometimes responsible for such other disease states as urticaria and various dermatoses, vasomotor rhinitis and vernal conjunctivitis, the exact nature and mechanism of production of hay fever and bronchial asthma are still unsettled questions. It may be stated as an established fact, however, that these diseases are not caused by any preformed toxins in the pollens, epidermal proteins, foods, dusts and other substances of animal and vegetable origin known to be etiologic agents. Since Wolf-Eisner,[1] in 1906, suggested that hay fever was caused by anaphylaxis, and Meltzer,[2] in 1910, suggested the same etiologic basis for bronchial asthma, it has been universally accepted that both are clinical expressions of what is called anaphylaxis, allergy or hypersensitiveness. But there has been and still is a large amount of controversial discussion and literature bearing not only upon the nature and mechanism of the processes involved but likewise upon the proper terminology.

I have elsewhere[3] given reasons for adopting von Pirquet's term "allergy" as a common designation for these diseases as a group because it so well expresses the fundamental change as being one of exaggerated susceptibility to the exciting substance; also for using the term "anaphylaxis" for those allergic diseases in which it is known that the exciting agent is a protein or a protein derivative, while reserving the term "hypersensitiveness" for those conditions, like the drug allergies, in which the exciting agent is apparently a nonprotein and nonantigenic agent. As will be shortly discussed with more detail, there are much clinical and some experimental data indicating that heredity plays an important rôle in the etiology of hay fever and bronchial asthma, and for this reason one may

accept Coca's suggestion for designating these two particular diseases and other natural allergies as "atopy" because of the inherited factor in their production. But as I hope to point out in this communication, the results of recent investigations have shown that in hay fever and bronchial asthma an antibody is present, that the exciting agents are known to be proteins or that the possibility of their protein nature has not been definitely excluded and that the processes are examples of anaphylaxis, the inherited factor being one of instability of the vasomotor system which greatly increases susceptibility to the effects of anaphylactic shock upon the muscle of blood vessels, which results in the production of congestion and edema as the characteristic lesions in human beings.

As just mentioned, the term anaphylaxis is applied to phenomena of this kind when the exciting agent is known to be of a protein nature. Not all of the known exciting agents of hay fever and asthma have been studied chemically, but Grove and Coca[4] have recently shown that the exciting agents in horse dander and green pea at least are digestible proteins, and I believe it is safe to assume that this applies to the epidermal products of mammals and fowls and to the foods in general. But as shown by Grove and Coca and also by Black,[5] the exciting agents in ragweed, house dust and orris root are apparently not digestible and nonprotein in nature. In other words, after the removal of proteins and other nitrogenous substances of these agents by tryptic digestion and dialysis, they were still capable of exciting positive skin reactions in susceptible individuals. In this respect they bear some resemblance to the exciting agent in tuberculin, which likewise has not given indisputable evidence of a protein nature. Also to the bacterial toxins, which, while actively antigenic, have not been definitely identified as proteins, although known to be destroyed by tryptic digestion and therefore differing in this respect from the exciting agent in the pollens. It may be that the latter possesses the chemical nature of the glucosides and in this respect resemble those polysaccharids isolated by Heidelberger and Avery[6] from pneumococci, which were found to be antigenic and similar to like substances isolated from yeasts, staphylococci and some other organisms. Since there is some immunologic evidence indicating that complex glucosides and

other foregn colloidal substances capable of stimulating body cells to activity may be hydrolyzed by enzymes with the production of antigenic substances, it is not improbable that the exciting agent in pollens and some other substances of vegetable origin may be glucosides, as likewise believed to be true of the exciting agent in poison ivy, sumac, etc., capable of being converted in the tissues into antigenic agents by enzymic hydrolysis or of uniting with plasma constituents with the production of a protein antigen. But aside from this possibility, there still exists the probability that the methods employed by Grove, Coca and Black for removing the nitrogen from the substances employed by them may have been insufficient for warranting the conclusion that the exciting agents were entirely protein free. Furthermore, as shown by Koessler, Parker, Walzer and Grove, pollen extracts, including those apparently free of protein, are now known to be antigenic and capable of sensitizing the guinea pig; similar results have also been observed with horse dander. It is true that this does not necessarily prove that the antigenic substance is a protein but it greatly strengthens this probability.

Another reason heretofore advanced for refusing to accept hay fever and bronchial asthma as anaphylactic phenomena has been a failure to demonstrate antibodies in the blood or cells and failure to passively transfer the susceptible state to other individuals by an injection of serum. But I have always maintained that failure to demonstrate antibodies and to passively transfer the state by injections of serum were no criteria, since failure in both attempts may be due to defective methods of study. The results of recent investigations have shown this to be true. Prausnitz and Küstner[7] have discovered that the intradermal injection of a small amount of serum from a fish-sensitive person into the skin of a nonsusceptible individual resulted in passively transferring the allergic state, as shown by the occurrence of positive skin reactions upon the application of fish extract to the sensitized skin. De Besche[8] has been able to confirm this work with the sera of asthmatics and various extracts, and Walzer has also obtained confirmatory evidence with some of the edible proteins. Not all normal human skins, however, can be sensitized in this way; but, according to Coca and Grove,[9] successful results have been ob-

served in about 89 per cent, and sensitization lasts at least four weeks. Furthermore, it has been apparently possible in some instances to sensitize the uteri of guinea pigs by injections of sera from hay fever and asthmatic persons, although experiments of this kind have frequently failed, and it is certain that human skins are more readily sensitized than the tissues of guinea pigs.

It is true that the sera of hay fever and asthmatic individuals may be and usually are unable to produce precipitation and complement fixation reactions in the test tube with extracts of the exciting agents, but successful results with both reactions have been reported, and negative results may be due to faulty technic, and especially in the case of the precipitin tests in which the quantitative relations between antibody and antigen must be very exact for positive reactions and concerning which we have little information in so far as these tests applied with the sera of hay fever and asthmatic individuals are concerned. However, the importance of this demonstration of precipitation and complement fixation in the test tube has been overemphasized. I admit that there is considerable evidence to show that the anaphylactic antibody and precipitin are identical, but failure to demonstrate precipitation in the test tube is by no means acceptable as evidence of the absence of anaphylactic antibody in the serum. Furthermore, it has been shown by Coca and his associates that the injection into human skins of antiegg and antipollen sera containing large amounts of precipitin did not result in sensitization, and the sera of hay fever and asthmatic individuals apparently free of precipitins are capable of not only sensitizing normal receptive human skins by the Prausnitz-Küstner method but likewise capable of neutralizing the exciting agent of some pollens and other substances in the test tube. As shown by Anderson and Schloss,[10] the blood of marantic infants may contain precipitins for cow's milk protein and yet show no clinical evidences of hypersensitiveness, although some may yield positive skin reactions; likewise in serum sensitization, the presence or absence of precipitin in the blood would appear to be without significance.

For these reasons at least, little or no significance is to be attached to the presence or absence or precipitins in relation

to the anaphylactic nature of hay fever and asthma. Coca and Grove have suggested calling the antibody in hay fever, asthma and other natural allergies "atopic reagin," but I can see no essential reason for this further addition to an already overburdened nomenclature, simply because the antibody in hay fever and asthma may not be identical with precipitin. Nor do I see any reason for assuming that the organs producing the antibody in hay fever and asthma and called "reaginogenic" by Coca and his associates are different from those producing the anaphylactic antibody; nor any reason for assuming that the hereditary influence in these diseases is due to the inheritance of a "reaginogenic organ" especially concerned in the production of a new or special antibody.

There is at least no doubt that an antibody is present in hay fever and bronchial asthma, and it is also agreed that the antibody is situated in the sensitized cells, where union with the antigen results in the production of the shock reaction constituting the attack of hay fever or asthma or the positive skin and mucous membrane reactions. It is certain that any antibody antigen reaction occurring in the body fluids is without injurious consequences. In other words, the humoral or anaphylatoxin theory has been abandoned in favor of the cellular theory of anaphylaxis, although we are totally ignorant of the nature and mechanism of the cellular reaction.

In so far as the human being is concerned, however, the "shock organ" responsible for hay fever and bronchial asthma would appear to be the smooth muscle of blood vessels. In the guinea pig and rabbit anaphylactic asthma may be due to tetanic contraction of the bronchial muscles, but I agree with Coca that in hay fever and asthma of human beings, as well as in urticaria, the production of congestion and edema are the essential and primary lesions, adrenalin constituting a physiologic antidote.

But how may one explain the influence of heredity upon hay fever and asthma? I am sure that all will agree with Cooke and Van der Veer,[11] Spain and Cooke,[12] regarding the familial nature of these and other natural anaphylactic states. Furthermore, as suggested by Lewis and Loomis, it may be that inbred guinea pigs inherit a greater tendency to acquired

sensitization than the majority of these animals. It is frequently possible to elicit a history of hay fever, asthma or some other anaphylactic state in one or both parents of hay fever and asthmatic individuals. Doerr believes that heredity influences the ability of the individual to produce anaphylactic antibody, and, as mentioned above, Coca and his associates believe that it is a question of familial transmission of a tissue capable of producing the "atopic reagin," which amounts almost to the same thing. But in my opinion, the influence of heredity is one involving the smooth muscle of blood vessels, and probably through the vasomotor nervous system. Hay fever and asthmatic individuals frequently show clinical evidences of vasomotor instability or irritability, and particularly to emotion and such physical agencies as changes in temperature and irritation of the skin. It is curious, but nevertheless true, in my experience, that colored people are less, very much less, likely to be victims of hay fever and asthma than whites. Indeed, I look upon hay fever and asthma as largely diseases of the whites, and especially of the well to do classes. I believe, therefore, that individuals inheriting this excitable or instable vasomotor system are particularly apt to suffer with hay fever and asthma, not because they are more easily sensitized, because the results of experiments indicate that they are not, nor because they have inherited a "reaginogenic organ," but because they are more susceptible to anaphylactic shocks by reason of this excitable condition of their blood vessels.

The question naturally arises whether or not human beings without hereditary predisposition ever acquire sensitization to the exciting agents of hay fever and bronchial asthma. Coca[13] states that not one of fifty individuals of his acquaintance employed in the collection of pollens acquired sensitization, but the frequency with which these diseases are observed in individuals of negative family histories indicates that sensitization may be acquired without hereditary predisposition, and especially when one remembers that both diseases, and especially hay fever, are quite uncommon among young children. The mere difficulty experienced in sensitizing guinea pigs and rabbits is not a criterion, although even guinea pigs have been sensitized by Dunbar,[14] Parker,[15] Alexander[16] and others, not only by injections of extracts of pollen and epidermal products

but even passively by injections of serum. This difficulty is only one relating to varying susceptibility of species which has long been recognized in anaphylactic investigation. It may be stated in passing, however, that hay fever and asthmatic individuals are said not to be more likely to acquire serum sensitization from injections of horse serum antitoxin than normal individuals, but, in my experience, they are sometimes more likely to react after intravenous injections of serum, and certainly their skins are more likely to react in a specific or nonspecific manner to protein solutions.

I believe, therefore, that Wolf-Eisner and Meltzer were correct in stating that hay fever and bronchial asthma are anaphylactic phenomena, although the former was wrong in explaining the mechanism on the basis of the production of a poison (called anaphylatoxin) by an interaction between pollen agent and antibody in the blood, with or without the activity of complement, since the cellular site of anaphylaxis has been so thoroughly supported by many investigators.

The positive skin and mucous membrane reactions, however, may be specific anaphylactic or nonspecific reactions, or a combination of the two. This is especially true of intradermal reactions, because of the added chances of producing trauma and eliciting physical reactions. It is much less likely with the dermal or cutaneous tests and these, therefore, are to be preferred for diagnostic purposes, although it is to be admitted that the intradermal tests are somewhat more sensitive, and especially with extracts of foods and bacteria. Not infrequently, however, one observes positive skin reactions to extracts of the pollens of plants to which it is certain that the patient has no access; also to foods which from the clinical standpoint do not appear to be harmful. These may be nonspecific and their nature obscure, but in the case of foods it is well established that anaphylactic sensitiveness may be present without clinical manifestations, and in the case of pollen extracts the reactions may be due to biologic relationship of the pollens or to a pure nonspecific reaction to the effects of trauma or other physical agents. Certainly there is no definite relation between the antibody content of the blood and the clinical severity of hay fever and bronchial asthma.

Finally, I believe that the specific treatment of hay fever and bronchial asthma by the subcutaneous injection of extracts of the exciting agent or agents is a process of true desensitization by which the anaphylactic antibody in sensitized cells is exhausted. According to McKenzie and Baldwin,[17] it is apparently possible to desensitize the nasal mucosa by the local application of pollen extracts, suggesting the local exhaustion of antibody in the sensitized cells of this organ. Cooke has questioned the correctness of this deduction and states that successful specific treatment of hay fever and asthma results in no noticeable diminution in the degree of sensitiveness of the skin or conjunctiva, and Coca states that he and Levine have shown that specific treatment does not lower the antibody (reagin) content of the blood. I have no experience to draw upon in so far as the antibody content of the blood is concerned, but I am sure that the successful treatment of hay fever with specific pollen extracts results in reducing and sometimes completely removing the positive skin reactions; in fact, I use this as a means for guiding the specific treatment of hay fever by desensitization, and especially in such matters as the dosage and number of injections to be given in preseasonal treatment. It is to be admitted, however, that hay fever and asthmatic individuals reaching this state of skin desensitization may still present clinical evidences of their anaphylactic state but not usually to the same degree as before desensitization.

SUMMARY.

In conclusion, I believe, therefore, that an analysis of recent investigations bearing upon the nature of hay fever and bronchial asthma warrants the statement that these allergic states are examples of anaphylaxis in which many of the subjects inherit an instability of the vasomotor system which renders them more susceptible to the effects of anaphylactic shock upon the smooth muscle of their blood vessels and resulting in the production of acute congestion and edema as the characteristic anaphylactic lesions of human beings. Also that these anaphylactic reactions occur in the involved cells by an unknown mechanism, and that the local or subcutaneous application of the exciting agent may result in the complete or par-

tial recovery from the anaphylactic state by the exhaustion of the anaphylactic antibody, properly designated as a process of desensitization.

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2101 PINE ST.

XLIX.

THE ALKALOIDS OF CEANOTHUS AMERICANUS AND THEIR USE IN THE CONTROL OF HEMORRHAGE IN LARYNGOLOGY.

By Richard Johnson Payne, M. D.,

St. Louis.

The increasing amount of surgery being performed by the otolaryngologist and the peculiar nature of the field in which the work is done offer ample opportunity for the observation of a drug reputed to control bleeding.

The difficulties which hemorrhage of the nose and throat present are too well defined to require elucidation. The location of the bleeding vessel and subsequent ligation are serious problems.

The peculiar structure of the tonsillar cavity increases the difficulty of locating the bleeding points and often conceals the nature of the bleeding. Capillary oozing is an annoying type of hemorrhage, which could be avoided by routine preoperative administration of a safe coagulant.

The work of Clark* on the chemistry of Ceanothus americanus and its alkaloids indicates several distinct advantages over other drugs now in general use as hemostatics. Remarkable depressions of clotting time have been reported† by the oral administration of a solution of the mixed alkaloids. I have used this solution in my work at the Missouri Baptist Sanitarium, the Barnes Hospital and various clinics for several months and have noticed that postoperative hemorrhage has been greatly reduced.

This observation led me to investigate the effect produced by oral administration of a marketed product‡ made from the

*Clark, A. H.: Am. Jour. Pharm., 98 (1926), 147.

†James T. Groot (work as yet unpublished).

‡The solution used in all of these cases is a preparation marketed by Flint, Eaton & Co., Decatur, Illinois, under the trade name "Ceanothyn."

extract of Ceanothus americanus and standardized to contain 1 mg. of the mixed alkaloids per cc. I have been unable to find any work in the literature covering the clinical observation of this drug as a coagulant. Its use as a hemostatic was suggested by Hammond*, and reference was found to the use of the drug as a "styptic for restraining hemorrhage from wounds."†

In all the cases described herein I have carefully followed this procedure:

1. Established normal coagulation time.
2. Administered solution of alkaloids, oral.
3. Observed coagulation time just before operation is performed.
4. Tonsillectomy under ethylene gas anesthesia (Sluder method).
5. Observed case for twelve hours following operation. Particular attention to tonsillar fossa.
5. The coagulation time was taken by the capillary tube method (technic is given below).

Technic: The finger is thoroughly cleansed with soap and water and dried with alcohol. A uniform puncture is made with a spring lancet (puncture should be deep enough to allow the blood to drop freely without pressure).

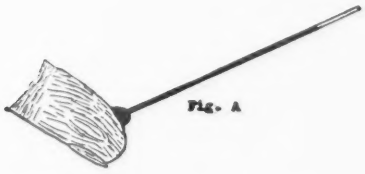
The first drop of blood is wiped away with sterile gauze, which should be free from lint. The second drop of blood is used for the test, and is drawn into the tube by capillarity. Fig. A.

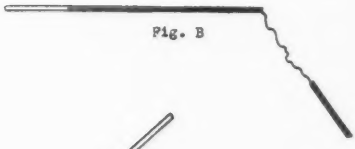
The tube should be about 10 cm. long and approximately 1 mm. in diameter. Attention must be given to the fact that the normal coagulation time and the coagulation time after administration of the drug be made with tubes of the same diameter.

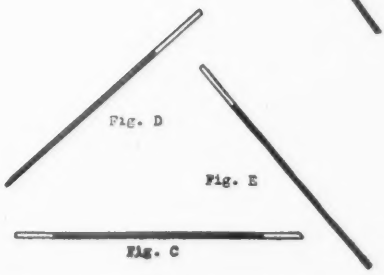
Immediately after the tube is filled it is laid on a porcelain rest and remains untouched for one minute. At fifteen second intervals thereafter the tube is picked up and a piece about 1 cm. long is broken off. When coagulation has taken place a thread of fibrin will connect the tube with the piece broken off.

*The Georgia Eclectic Medical Journal, Atlanta, Georgia, 1879-8-, 1, 355-359.

†The Boston Medical and Surgical Journal (1836), XV, p. 42.







The appearance of this fibrin thread indicates the coagulation time. Fig. B.

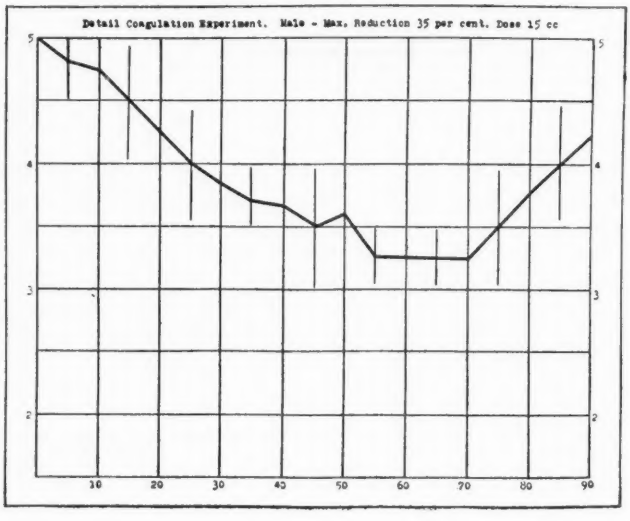
These tests may be carried out at ordinary room temperature with uniform results, providing this technic is carefully observed.

Note: Indication of the clotting time can be obtained by tilting the tube to allow the blood to flow from one end of the tube toward the other. So long as the blood flows freely in the tube coagulation has not taken place, and breaking the tube is unnecessary. See Figs. C, D, E.

CLINICAL OBSERVATION.

Effect on Coagulation Time.—My interest in the hemostatic properties of this drug was enhanced by a study of Groot's work (as yet unpublished), in which he has shown "the true

contour of the graph representing the actual occurrence of coagulation events." A single experiment (normal subject) of this type, in which I have endeavored to determine the rapidity with which the coagulation time is depressed and the length of time this depression is apparent, is outlined below. Coagulation tests were made at five minute intervals following the oral administration of the solution. This experiment indicates that an appreciable reduction in clotting time is effected within ten minutes after the oral administration of a 15 cc. dose of the solution. This decline continues for about forty minutes, is sustained at the point of maximum depression for another twenty minutes, after which time there is a gradual rise. The coagulation tests were continued for ninety minutes after the solution was given. At this point the clotting time was fifty seconds shorter than normal. Estimating a continuation of the rise on this basis, the return to normal would be complete in approximately two hours. While it was impossible to make such detailed tests on the subjects used in these experiments, the general results would indicate that coagulation events followed very closely the line of this experiment.



Following the plan of administration and observation which I have outlined, I have observed the effects on coagulation in 234 cases. These cases have been grouped according to sex, age, and dose administered. In all of these cases the solution was given preoperatively on an empty stomach.

MALE, AGES ONE TO TEN YEARS, 15 CC. DOSE.

The first group tabulated here shows the effect on coagulation produced by single 15 cc. doses of the solution orally administered to male patients, ranging in age from one to ten years.

This group includes eighty cases whose average normal coagulation time was 3.75 minutes. The average time elapsed after the administration of the solution was fifty-three minutes, and the average reduction in coagulation time was 21.0 per cent. This tabulation shows the effect produced in the individual cases:

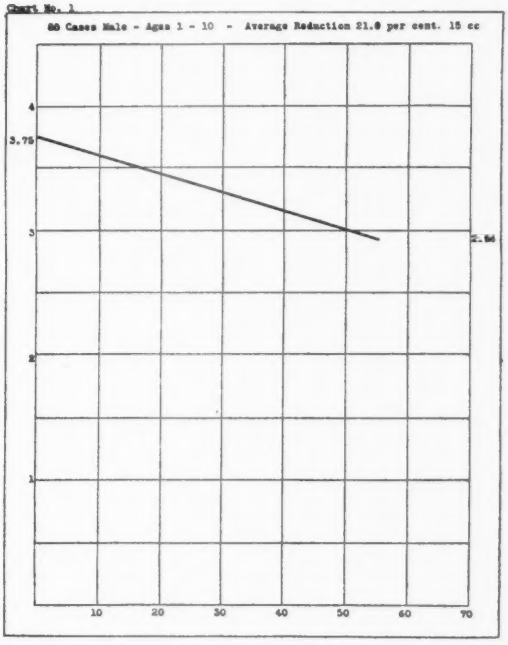
TABLE 1.

No.	Case	Age	Sex	C. T. Normal	C. T. Depressed	Elapsed Time	Dose
1	F. V.	9	M	4.30	3.30	25	15 cc.
2	L. R.	7	M	3.00	2.30	30	15 "
3	J. D.	5	M	3.00	2.00	30	15 "
4	W. C.	8	M	3.15	2.30	30	15 "
5	P. L.	6	M	3.15	2.30	30	15 "
6	C. H.	4	M	2.15	1.45	30	15 "
7	F. V.	8	M	3.00	2.45	30	15 "
8	E. C.	9	M	4.15	3.45	30	15 "
9	B. D.	7	M	3.45	3.15	30	15 "
10	B. W.	9	M	4.30	3.00	30	15 "
11	B. W.	9	M	3.15	2.30	35	15 "
12	H. H.	4	M	3.00	2.15	35	15 "
13	W. A.	9	M	3.15	2.30	35	15 "
14	C. L.	8	M	3.30	3.15	35	15 "
15	M. K.	8	M	3.30	3.15	40	15 "
16	D. D.	5	M	3.30	2.30	40	15 "
17	B. M.	6	M	5.15	4.15	40	15 "
18	S. C.	9	M	3.00	2.30	40	15 "
19	N. K.	7	M	4.15	3.45	40	15 "
20	M. H.	6	M	3.30	3.00	40	15 "
21	R. S.	9	M	4.15	3.45	40	15 "
22	T. J.	7	M	4.15	3.45	40	15 "
23	G. M.	5	M	4.30	4.00	40	15 "
24	G. H.	7	M	3.30	3.00	40	15 "
25	S. E.	9	M	3.30	3.00	40	15 "
26	K. B.	8	M	2.45	2.00	45	15 "
27	J. M.	9	M	5.30	4.15	45	15 "
28	R. Z.	8	M	3.30	3.00	45	15 "
29	J. B.	8	M	4.00	2.15	45	15 "
30	C. M.	8	M	4.00	2.00	45	15 "

TABLE 1.—Continued.

No.	Case	Age	Sex	C. T. Normal	C. T. Depressed	Elapsed Time	Dose
31	C. J.	4	M	3.15	2.15	45	15 "
32	C. J.	4	M	3.00	2.15	45	15 "
33	M. H.	9	M	4.00	1.30	45	15 "
34	F. M.	7	M	3.30	3.00	50	15 "
35	D. W.	8	M	4.00	3.00	50	15 "
36	J. S.	8	M	4.00	3.00	50	15 "
37	O. H.	2	M	3.30	2.45	50	15 "
38	J. S.	8	M	3.00	2.15	50	15 "
39	J. S.	5	M	3.15	2.15	50	15 "
40	L. W.	9	M	3.00	1.45	50	15 "
41	A. B.	10	M	5.15	3.30	50	15 "
42	G. F.	9	M	6.00	4.30	50	15 "
43	C. A.	5	M	3.15	2.30	50	15 "
44	D. J.	9	M	5.15	4.15	50	15 "
45	I. H.	8	M	3.45	3.00	50	15 "
46	D. S.	7	M	3.45	3.00	50	15 "
47	P. C.	6	M	3.45	3.00	50	15 "
48	S. G.	6	M	4.15	2.30	55	15 "
49	P. W.	9	M	5.30	4.15	60	15 "
50	R. H.	9	M	3.30	3.00	60	15 "
51	A. H.	4	M	4.00	3.30	60	15 "
52	J. G.	10	M	3.15	2.45	60	15 "
53	R. B.	9	M	4.00	2.30	60	15 "
54	W. W.	9	M	3.45	2.45	60	15 "
55	H. O.	8	M	3.15	2.45	60	15 "
56	A. W.	7	M	3.15	2.45	60	15 "
57	W. D.	10	M	3.45	2.45	60	15 "
58	H. K.	9	M	4.15	3.45	60	15 "
59	M. P.	10	M	4.15	2.45	60	15 "
60	J. G.	7	M	4.15	3.15	60	15 "
61	L. B.	10	M	4.15	4.00	60	15 "
62	J. H.	9	M	4.00	3.15	65	15 "
63	T. C.	9	M	4.45	3.00	65	15 "
64	L. P.	8	M	2.45	2.30	65	15 "
65	P. P.	9	M	3.30	2.30	65	15 "
66	A. S.	8	M	3.15	3.00	65	15 "
67	W. S.	8	M	2.45	2.30	65	15 "
68	M. T.	4	M	3.00	2.30	70	15 "
69	P. F.	10	M	4.00	3.45	70	15 "
70	P. C.	7	M	3.15	3.00	70	15 "
71	B. E.	8	M	3.00	2.30	70	15 "
72	M. M.	9	M	4.30	3.00	80	15 "
73	J. D.	8	M	3.15	2.45	80	15 "
74	B. Y.	5	M	4.15	4.00	85	15 "
75	J. B.	5	M	3.45	3.30	90	15 "
76	R. P.	7	M	3.15	3.00	90	15 "
77	O. L.	9	M	3.30	3.15	90	15 "
78	J. J.	5	M	3.30	3.00	90	15 "
79	G. H.	4	M	5.00	4.30	95	15 "
80	J. H.	4	M	3.15	2.30	95	15 "
Average				3.75	2.96	53	

Shown graphically, this average reduction in coagulation time would be represented by a straight descending line, as shown below.



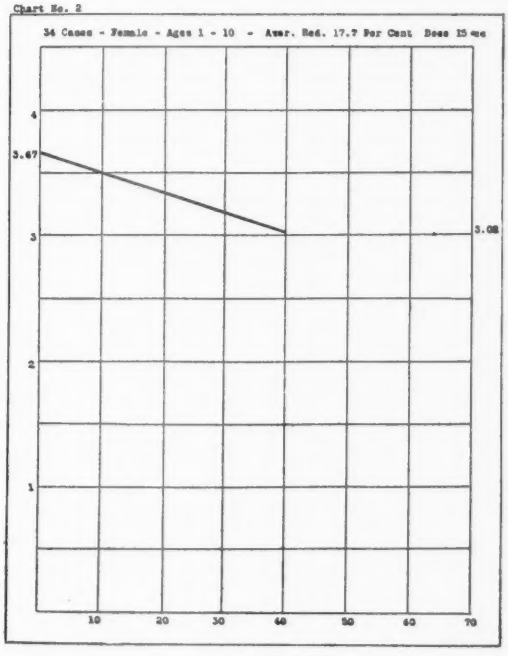
FEMALE, AGES ONE TO TEN YEARS, 15 CC. DOSE.

Another series of experiments is grouped here showing the effect on coagulation time following the oral administration of the solution to female patients between the ages of one and ten years. A single 15 cc. dose was administered orally to each patient and observed as in the previous group. This series includes thirty-four cases with an average normal coagulation time of 3.67 minutes, and shows an average depression in clotting time of 17.7 per cent in forty-two minutes after the administration. Individual results are shown in this tabulation:

TABLE 2.

No.	Case	Age	Sex	C. T. Normal	C. T. Depressed	Elapsed Time	Dose
1	F. B.	8	F	3.15	3.00	20	15 cc.
2	D. A.	7	F	3.30	3.00	30	15 "
3	J. T.	10	F	3.30	3.15	30	15 "
4	J. E.	10	F	3.00	2.45	30	15 "
5	C. G.	6	F	4.00	2.45	35	15 "
6	C. G.	7	F	3.30	2.30	35	15 "
7	J. C.	6	F	3.30	2.30	40	15 "
8	D. H.	9	F	4.00	3.15	40	15 "
9	R. H.	8	F	3.30	2.30	40	15 "
10	F. S.	6	F	3.30	2.30	40	15 "
11	N. T.	7	F	5.15	4.45	40	15 "
12	J. C.	10	F	3.15	3.00	40	15 "
13	F. P.	6	F	3.00	2.45	40	15 "
14	N. G.	7	F	3.00	2.45	40	15 "
15	L. M.	10	F	4.30	2.45	45	15 "
16	S. H.	5	F	4.15	3.45	45	15 "
17	H. D.	4	F	3.00	1.15	45	15 "
18	L. S.	9	F	2.45	2.30	50	15 "
19	G. W.	9	F	3.15	2.30	50	15 "
20	A. M.	9	F	4.15	3.45	50	15 "
21	B. C.	5	F	4.00	3.45	50	15 "
22	S. M.	9	F	3.30	3.00	50	15 "
23	C. G.	6	F	5.00	4.30	50	15 "
24	D. H.	4	F	4.00	3.30	50	15 "
25	F. H.	8	F	3.15	2.00	55	15 "
26	D. H.	8	F	5.00	4.30	55	15 "
27	O. K.	8	F	4.00	2.30	60	15 "
28	C. Mc.	8	F	3.15	2.45	60	15 "
29	D. L.	10	F	4.00	3.15	60	15 "
30	A. C.	8	F	4.15	4.00	60	15 "
31	F. F.	9	F	3.00	2.45	60	15 "
32	F. T.	9	F	4.00	3.15	60	15 "
33	F. S.	9	F	3.00	2.30	90	15 "
34	D. C.	4	F	3.00	2.45	90	15 "
Average				3.67	3.02	42	

Graphically, this average depression may be illustrated as shown below.



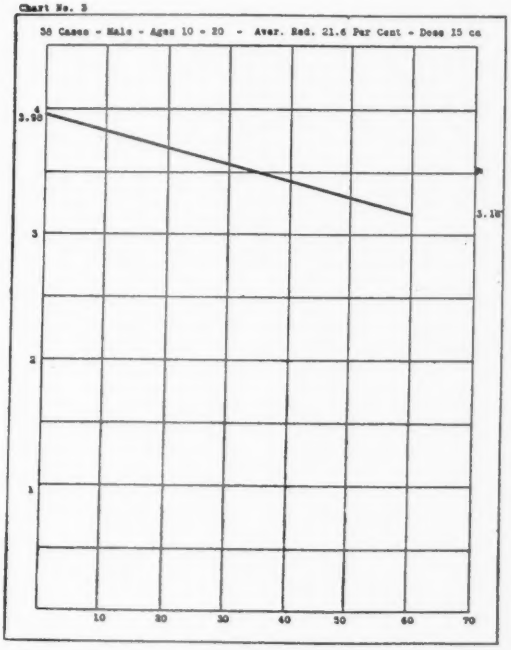
MALE, AGES TEN TO TWENTY YEARS, 15 CC. DOSE.

Effects on coagulation time produced by the oral administration of the solution to thirty-eight male subjects, ages ranging from ten to twenty years, are grouped here. A single 15 cc. dose was given to each patient preoperatively, and the result observed as before. The average normal clotting time of 3.98 minutes was shortened to 3.16 minutes, a reduction of 21.6 per cent in an average of sixty minutes after the solution was given. Individual results are tabulated here.

TABLE 3.

No.	Case	Age	Sex	C. T. Normal	C. T. Depressed	Elapsed Time	Dose
1	H. S.	12	M	2.45	2.15	20	15 cc.
2	G. S.	11	M	4.00	3.45	30	15 "
3	F. T.	13	M	4.30	3.15	35	15 "
4	D. O.	12	M	3.45	2.30	40	15 "
5	J. S.	12	M	3.45	3.00	40	15 "
6	E. P.	13	M	3.00	2.30	40	15 "
7	L. D.	14	M	3.15	2.15	45	15 "
8	J. S.	14	M	4.00	2.30	45	15 "
9	R. S.	13	M	4.15	3.15	45	15 "
10	F. C.	12	M	3.30	1.30	45	15 "
11	T. M.	11	M	4.00	3.30	45	15 "
12	C. P.	12	M	4.15	2.45	45	15 "
13	N. B.	11	M	5.15	4.30	50	15 "
14	H. G.	13	M	5.15	4.15	50	15 "
15	H. B.	11	M	4.15	3.15	50	15 "
16	P. O.	14	M	4.00	3.15	60	15 "
17	J. B.	17	M	3.45	2.45	60	15 "
18	M. H.	20	M	7.00	4.30	60	15 "
19	C. P.	14	M	3.30	3.15	60	15 "
20	R. R.	17	M	4.00	3.30	60	15 "
21	J. L.	11	M	4.30	3.45	60	15 "
22	S. M.	11	M	4.45	4.15	60	15 "
23	T. M.	11	M	3.45	3.15	60	15 "
24	E. M.	13	M	3.30	3.15	60	15 "
25	C. W.	12	M	3.00	2.15	65	15 "
26	C. R.	12	M	3.30	2.30	65	15 "
27	J. H.	15	M	3.30	2.30	70	15 "
28	E. M.	12	M	3.30	2.30	70	15 "
29	V. Z.	12	M	4.15	4.00	70	15 "
30	C. Z.	13	M	3.30	3.00	70	15 "
31	R. S.	15	M	4.00	2.45	80	15 "
32	B. S.	13	M	4.00	3.15	80	15 "
33	W. M.	13	M	4.30	4.15	80	15 "
34	H. S.	15	M	4.00	3.45	85	15 "
35	P. L.	14	M	4.30	4.00	90	15 "
36	J. W.	13	M	3.45	3.00	90	15 "
37	J. D.	12	M	3.30	3.00	95	15 "
38	H. T.	11	M	3.30	2.45	100	15 "
Average				3.98	3.16	60	

This average reduction is shown graphically here.



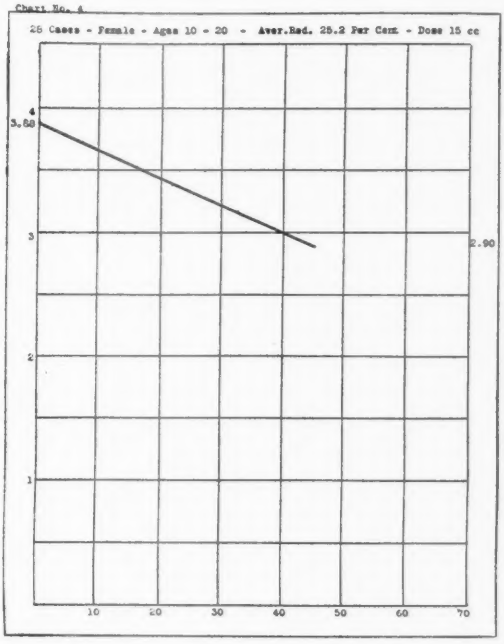
FEMALE, AGES TEN TO TWENTY YEARS, 15 CC. DOSE.

Results observed following the oral administration of single 15 cc. doses of the solution to twenty-eight female subjects show an average depression in clotting time of 25.2 per cent, in an average of forty-six minutes after the administration. This depression is somewhat greater than that produced by similar dosage on male subjects in the same age range. The explanation probably lies in the fact that the depressed clotting time was observed at a point nearer the actual maximum depression than in the previous group. The tabulated data given here shows the effects produced in the individual cases:

TABLE 4.

No.	Case	Age	Sex	C. T. Normal	C. T. Depressed	Elapsed Time	Dose
1	A. C.	15	F	3.15	3.00	15	15 cc.
2	J. C.	16	F	5.00	3.00	30	15 "
3	C. C.	11	F	3.00	2.00	30	15 "
4	G. C.	12	F	2.45	2.30	30	15 "
5	H. J.	11	F	4.15	3.45	30	15 "
6	S. S.	12	F	3.45	3.15	30	15 "
7	B. G.	18	F	3.30	3.15	35	15 "
8	M. L.	13	F	2.45	2.00	35	15 "
9	M. M.	16	F	4.30	2.45	40	15 "
10	O. H.	15	F	3.15	2.00	40	15 "
11	V. H.	16	F	3.15	2.30	40	15 "
12	S. L.	15	F	4.00	1.15	45	15 "
13	D. E.	20	F	5.30	4.15	45	15 "
14	M. P.	17	F	5.00	3.30	45	15 "
15	K. J.	13	F	3.45	3.15	45	15 "
16	D. M.	18	F	5.00	3.15	50	15 "
17	V. R.	11	F	3.45	2.45	50	15 "
18	V. G.	11	F	4.15	2.30	60	15 "
19	J. J.	14	F	3.15	2.15	60	15 "
20	C. V.	20	F	3.30	2.45	60	15 "
21	V. F.	11	F	3.00	2.15	60	15 "
22	P. C.	15	F	5.15	3.30	60	15 "
23	M. B.	20	F	4.00	3.15	60	15 "
24	M. M.	18	F	4.00	3.45	60	15 "
25	E. M.	12	F	3.45	3.30	60	15 "
26	M. H.	14	F	3.00	2.30	60	15 "
27	N. S.	12	F	3.30	2.45	60	15 "
28	E. R.	15	F	5.00	4.00	60	15 "
Average				3.88	2.90	46	

Graphically, this average depression is illustrated below.



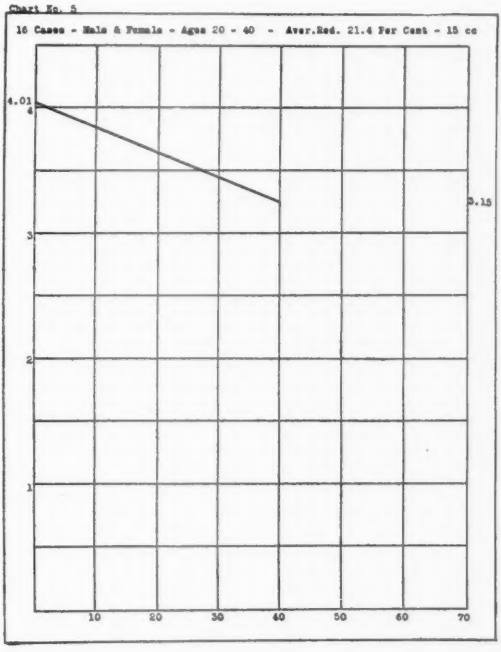
MALE AND FEMALE, AGES TWENTY TO FORTY YEARS, 15 CC. DOSE.

Coagulation time of sixteen subjects, of both sexes, whose ages range from twenty to forty years, was depressed 21.4 per cent following the oral administration of single 15 cc. doses of the solution in an average of thirty-eight minutes. This average depression corresponds, for the dosage given, very closely to results observed in the preceding four groups and indicates that sex or age does not influence the results obtained. The following tabulation shows the individual results observed:

TABLE 5.

No.	Case	Age	Sex	C. T. Normal	C. T. Depressed	Elapsed Time	Dose
1	M. B.	26	M	4.00	3.45	15	15 cc.
2	O. F.	34	M	3.00	2.45	15	15 "
3	G. H.	47	M	2.45	2.30	15	15 "
4	L. H.	22	M	3.30	2.15	15	15 "
5	G. M.	40	M	3.15	3.00	15	15 "
6	T. S.	40	F	3.30	3.00	30	15 "
7	B. T.	37	F	5.15	4.00	30	15 "
8	M. L.	28	F	4.30	3.45	40	15 "
9	H. L.	28	F	3.30	2.30	45	15 "
10	A. K.	27	M	5.30	4.00	45	15 "
11	A. M.	28	F	6.00	3.00	45	15 "
12	A. B.	24	F	3.00	2.15	60	15 "
13	H. H.	33	F	3.30	3.00	60	15 "
14	G. M.	30	M	4.30	4.00	60	15 "
15	S. A.	35	M	4.30	4.00	60	15 "
16	L. A.	24	F	4.00	2.45	70	15 "
Average				4.01	3.15	38	

The chart given below shows graphically the average results obtained in this group of observations:



MALE AND FEMALE, AGES ONE TO TWENTY, 30 CC. DOSE.

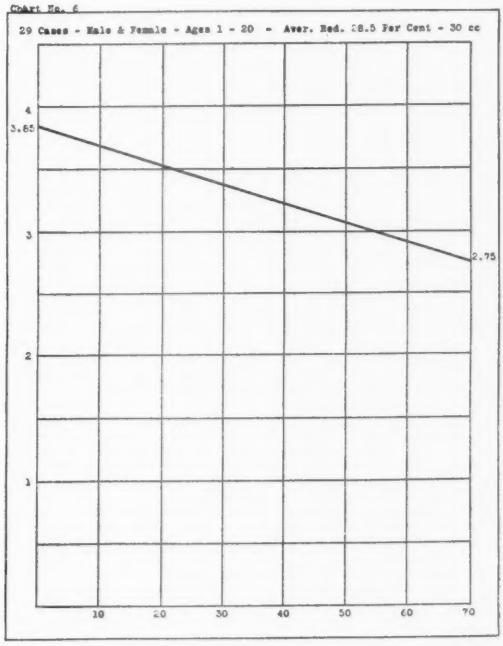
Twenty-nine cases were observed following the oral administration of single 30 cc. doses to subjects whose ages range from one to twenty years. An average depression in clotting time of 28.5 per cent was observed seventy minutes following the administration. This depression is somewhat greater than that produced by 15 cc. doses, although it is quite probable that the reduction was not observed at the point of maximum depression. Groot observes that the maximum depression is reached between twenty and forty minutes after 30 cc. doses of

the drug are given. Tabulation of the individual results obtained are given here. No variation in results is observed that would indicate an influence of sex or age:

TABLE 6.

No.	Case	Age	Sex	C. T. Normal	C. T. Depressed	Elapsed Time	Dose
1	L. M.	10	M	3.30	3.15	15	30 cc.
2	J. B.	5	M	3.30	3.00	25	30 "
3	G. A.	6	M	3.00	2.30	35	30 "
4	T. T.	4	M	3.00	2.15	45	30 "
5	S. M.	15	M	5.30	2.45	50	30 "
6	L. G.	18	F	3.45	2.45	50	30 "
7	R. M.	10	M	4.30	2.45	55	30 "
8	J. R.	9	M	4.15	2.15	60	30 "
9	L. C.	4	F	3.45	2.00	60	30 "
10	E. H.	9	F	3.45	2.45	60	30 "
11	W. M.	4	M	3.45	2.30	65	30 "
12	W. R.	6	M	3.15	3.00	65	30 "
13	D. L.	19	F	4.00	2.30	65	30 "
14	E. M.	7	M	3.45	2.30	70	30 "
15	W. I.	8	M	4.00	3.15	70	30 "
16	U. L.	8	M	3.15	3.00	70	30 "
17	F. T.	5	F	4.00	2.45	70	30 "
18	R. P.	8	M	3.15	2.30	75	30 "
19	C. M.	4	M	3.30	2.15	80	30 "
20	I. M.	8	M	3.45	3.00	85	30 "
21	C. K.	8	M	3.30	2.30	90	30 "
22	J. S.	9	M	4.00	3.00	90	30 "
23	H. S.	8	M	4.30	3.30	90	30 "
24	M. M.	6	M	3.15	3.00	90	30 "
25	D. W.	8	M	3.15	3.00	90	30 "
26	C. C.	10	M	4.30	3.30	90	30 "
27	C. F.	8	M	4.30	2.45	95	30 "
28	E. M.	4	M	3.45	2.15	100	30 "
29	B. A.	17	M	5.30	3.00	120	30 "
Average				3.85	2.75	70	

The average result is shown graphically below:



RECAPITULATION.

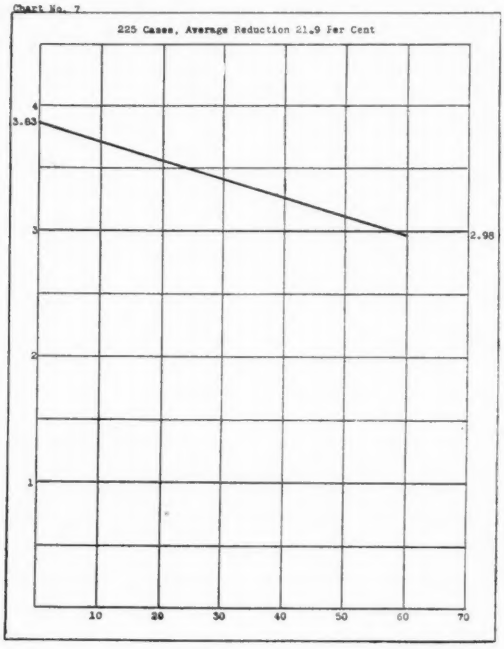
A summary of the data on these six groups is given below. A study of this comparative data verifies the opinion expressed before that results are not influenced by age or sex, but are directly influenced by the elapsed time after administration and dosage given.

It would appear from the data presented here that best results are to be obtained in preoperative use by administering the solution about forty-five minutes prior to the operation:

TABLE 7.

Table Number	Number of Cases	Sex	Age Range	Dose	Average Elapsed Time	Per cent Reduction
1	80	M	1—10	15 cc.	53	21.0
2	34	F	1—10	15 cc.	42	17.7
3	38	M	10—20	15 cc.	60	21.6
4	28	F	10—20	15 cc.	46	25.2
5	16	M & F	20—40	15 cc.	38	21.4
6	29	M & F	1—20	30 cc.	70	28.5
Average Reduction in 225 Cases						21.9

An average depression in coagulation time of 21.9 per cent was produced by the dosage mentioned. A composite average graph showing this reduction in clotting time in these 225 cases may be shown as follows:



The cases which have been shown in these group tabulations presented no unusual difficulties, and in only a few instances could the clotting time be considered as above normal. However, I have encountered several cases in my work in which the coagulation times were excessively high, and unusual reductions were obtained by administration of the solution. They are described briefly here:

M. M., male, 47 years. Repeated attacks of epistaxis. Admitted to hospital on four previous occasions, practically exsanguinated. History of hemophilia. Clotting time, 11 minutes. Two 1 ounce doses Ceanothyn administered at 30 minute intervals. Clotting time reduced to five minutes within an hour. Submucous resection of nasal septum was performed. Practically no bleeding.

M. C., female, 10 years. Clotting time, 12 minutes. Solution administered in single dose, ¾ ounce. Clotting time reduced to five minutes in one hour. Tonsillectomy performed. No postoperative hemorrhage.

J. B., male, 22 years. History of hemophilia. Practically exsanguinated following tooth extraction at previous date. Clotting time, nine and one-half minutes. Administered solution in single 1 ounce dose. One hour later the coagulation time was five minutes. One tonsil removed. No hemorrhage. Eleven days later clotting time was ten minutes. Two doses of ¾ ounce each of Ceanothyn were administered preceding removal of the other tonsil. Clotting time again reduced to five minutes. No postoperative hemorrhage.

A. J., female, 14 years. Coagulation time, 8¾ minutes. History of hemophilia. Administered Ceanothyn in ¾ ounce dose. Clotting time reduced to 4¾ minutes in one hour. Tonsillectomy. No postoperative bleeding.

P. M., male, 9 years. Clotting time, 7¾ minutes. No history of hemophilia. Administered 1 ounce of solution. Clotting time reduced to 3½ minutes. Tonsillectomy with practically no bleeding.

A. T., male, 18 years. Adenoids removed two years previously, at which time very severe postoperative hemorrhage occurred. Postnasal and anterior nasal packs were inserted, but patient continued to bleed for four days at intervals. Otitis media as complication. Recovery after three weeks'

stormy course. Patient readmitted to hospital for chronic suppurative right antrum. Clotting time, 11½ minutes. A 1 ounce dose of Ceanothyn administered. Clotting time reduced to 6 minutes. Intranasal antrum operation. Only usual amount of bleeding on removal of nasal packs.

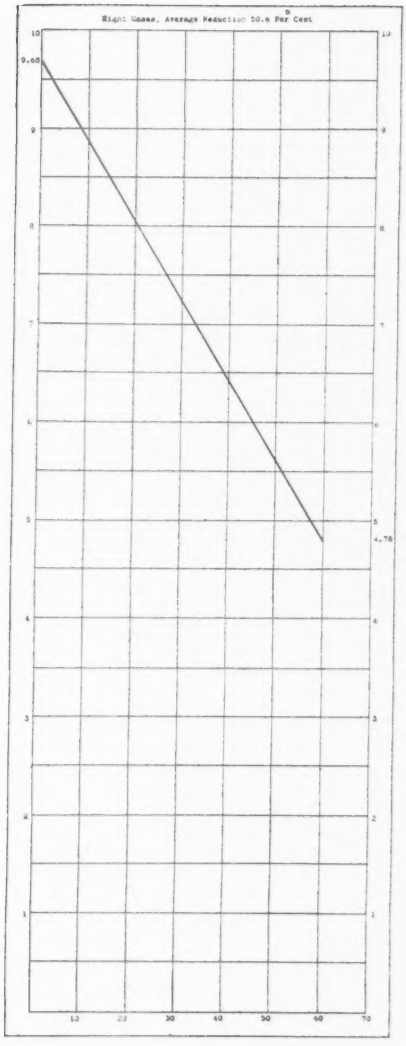
Dr. Scott Heuer, St. Louis, has reported one case to me, quite briefly, as follows:

"Male, 45 years. Intractable epistaxis. Could not be controlled with adrenalin, etc. Administration of this alkaloid solution, ½ ounce dose, controlled bleeding within 30 minutes."

Dr. F. C. Howard, St. Louis, also describes an interesting case which he has recently observed. He says:

"Mrs. W. B., 40 years. Entered St. John's Hospital three months ago. No history of hemophilia. Clotting time, 7 minutes. Administration of Ceanothyn, ½ ounce. Clotting time reduced within an hour to 4 minutes. Tonsillectomy performed. No postoperative hemorrhage."

In these cases an average reduction in clotting time of 50.6 per cent was effected by the oral administration of the alkaloid solution, approximately one hour after the drug was given. The average depression in this special group of cases may be shown graphically as follows:


Eight Cases, Average Reduction 50.6 Per Cent
9.68
4.78
10
9
8
7
6
5
4
3
2
1
10
20
30
40
50
60
70

SUMMARY.

The use of this standard solution presents several distinct advantages to the nose and throat surgeon which may be summarized as follows:

There was a consistent depression in coagulation time following the oral administration of the solution.

This depression in coagulation time amounts to an average of 21.9 per cent in these 225 cases.

Coagulation will be hastened by the administration of this solution, even in cases where the normal is low.

Depression of coagulation time is apparent within fifteen minutes after the administration of the solution, and this depression reaches its maximum about forty-five minutes after the drug is given.

The effect on coagulation time is not influenced by the age or sex of the patient.

Coagulation time is depressed more rapidly and to a greater extent in those cases in which the normal clotting time is high. Eight pathologic cases in which the average normal coagulation time was 9.68 minutes showed an average reduction in clotting time of 50.6 per cent within one hour following the administration of the solution.

There was practically no postoperative bleeding in these cases.

Adjunctive treatment was unnecessary. The simple oral administration of a single dose of the solution was sufficient to produce the results described.

Acknowledgment is made to Guy C. Taylor, who very kindly gave his time and counsel in advising the administration of the preparation used in these experiments, and for his assistance in demonstrating the technic used in determining the coagulation time.

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L.

LATERAL SINUS THROMBOSIS WITH REPORT OF FIVE CASES.

By E. F. Chase, M. D.,

Seattle.

One of the common, dreaded and frequently unavoidable complications of infection of the middle ear and mastoid process is an extension of the infection to the sigmoid and horizontal portion of the lateral sinus, causing a phlebitis, which is often followed by a partial or complete thrombosis, with its characteristic syndrome and sequelæ.

Thrombosis of the lateral sinus may be designated as primary and secondary. Primary lateral, as well as other sinus thrombosis, occurs in children under six years of age with chronic diarrhea and marasmus,[1] and is not an uncommon cause of infantile hemiplegia. It is also observed in chlorosis, anemia and the terminal stages of wasting diseases, as in cancer and phthisis. Secondary lateral, and other sinus thrombosis, follows septic lesions about the head, which drain into the immediate or collateral circulation of the lateral sinus and induce phlebitis and thrombosis, as in boils, carbuncles, sinusitis, parotitis, orbital cellulitis and in carious teeth, peritonsillar and pharyngeal abscesses.

In this paper the author considers only those cases of lateral sinus thrombosis that are of otitic origin.

How frequently this pathologic process occurs is difficult to ascertain, but, from available statistics, we are led to believe that sinus thrombosis occurs in suppurative mastoiditis in from 3 to 6 per cent of the operative cases, the incidence depending, no doubt, upon the virulence of the infecting organism during any stated period or epidemic.

Naftzger[2] reports 6 cases in 192 (3.12 per cent) and quotes Hill as having 7 cases in 166 (4.21 per cent); Downey, 5 cases in 79 (6.32 per cent); Gerber, 25 cases in 524 (4.77 per cent); Welty, 3 cases in 100 (3.33 per cent), and the Massachusetts Charitable Eye and Ear Infirmary, 19 cases in 497 (3.82 per

cent). To these may be added statistics from the Mayo Clinic,[3] which show that during a period of five years they encountered 19 cases in over 500 (3.8 per cent). The statistics as quoted above give an average total of 4.34 per cent.

That we may have a proper conception of the process of extension of infection from the middle ear and mastoid cells to the adjacent structures, we take the liberty of giving a brief review of the gross anatomy of this region.

The lateral sinus, for the purpose of description, is considered as having a transverse and a vertical portion. The former extends from the internal occipital protuberance to the posterior inferior angle of the parietal bone. The latter extends from the posterior inferior angle of the parietal bone along the sigmoid groove in the bone, to the jugular bulb, and is known as the sigmoid portion of the sinus. The right lateral sinus is continuous with the superior longitudinal sinus and is larger than the left, and its relationship to the mastoid cells is such that it is rendered more liable to infection than the left in the proportion of 3 to 2.[4] The left lateral sinus is smaller and is continuous with the straight sinus. There is usually a communication between the right and left lateral sinuses, either through a small communicating branch or through a fenestra in their walls.

The lateral sinus receives contributing veins as follows: (1) The mastoid emissary vein with numerous other small veins from within the immediate vicinity, (2) condyloid emissary veins, (3) superior petrosal sinus with venules from the temporosphenoidal lobe, (4) inferior cerebral and inferior cerebellar veins, and (5) diploic veins.

That portion of the lateral sinus which corresponds to, and lies within the groove in the temporal bone, is designated as the sigmoid sinus, and the angle at the junction of the vertical and horizontal portions as the knee of the sinus.

The knee is the portion of the lateral sinus which is most commonly attacked. The next most common site to become involved in an infectious process is the jugular bulb. The jugular bulb, supported and protected by the jugular fossa, is a downward continuation and expansion of the vertical portion of the lateral sinus just before it emerges into the internal jugular vein.

It is to be noted that the jugular bulb is in venous connection with (1) the cavernous sinus by the inferior petrosal, (2) the internal ear by the vena aqueducti cochleæ, (3) the venous plexus of the cerebellar fossa, (4) the occiptal sinus, (5) the spinal plexuses of the spinal canal by the vena condyloidæ. Thus it is observed, as has been pointed out by others, that infection can be distributed with apparent facility to the internal jugular, the central nervous system and the various sinuses and plexuses of the cranium and spinal canal, because of their close proximity to the middle ear and mastoid. We would point out, at this time, what is probably equally true, that the free and unrestricted anastomoses in this region act to safeguard and conserve the body economy by permitting changes in the circulation sufficient to sustain physiologic function and repair in case of sinus thrombosis.

The actual invasion of the sinus may occur in one of two ways: (1) by continuity, (2) by metastasis. Invasion by continuity consists in a process of infective cellular absorption and destruction of the sinus plate, with the formation of a perisinus abscess and phlebitis, and an associated thrombus resulting therefrom. The second route, or infection by metastasis, is exhibited by involvement of the lateral sinus following a mild middle ear infection, or a furunculosis of the external auditory canal, without gross evidence of an involvement of the mastoid cells.

Infection of the lateral sinus is caused by the giving way, or breaking down, of Nature's protective barrier, due to the virulence of the invading organism or to the lack of resistance upon the part of the patient, or both.

The pathology of sinus thrombosis is described as a periphlebitis which progresses and extends to the various layers of the vessel wall until a true phlebitis results, with an involvement of the intima, which favors the formation of a clot within its lumen. The entire lumen may be obstructed with the thrombus extending up to or beyond the torcular herophili and downward to the subclavian vein, involving all communicating sinuses within this area. Campbell[4] tersely describes the formation of a thrombus as: "At first there is a phlebitis—an infection of the tissue making the wall of the vein; then comes a breach in the inner lining of the wall, a slowing of the blood

current and formation of a coagulum. This coagulum becomes infected and a thrombus is formed and the way opened to a general bacteremia. The coagulum grows until a mural clot is formed, and particles of this may be detached by the blood current and thrown into the general circulation, causing symptoms of bacteremia."

The sinus may be completely obliterated by organization of the thrombus, or the sinus may undergo recanalization and the circulation be restored, or the infected thrombus may disintegrate and rupture into the mastoid wound or into the cranial cavity, causing a cerebellar abscess or a purulent meningitis.

According to Braun,[5] the newly formed thrombi contain few, if any, bacteria, which, if true, gives the inference that the thrombus is evolved and later invaded, in the majority of cases, by the streptococcus hemolyticus, as this is the organism usually recovered. Lillie[6] reports a case in which a staphylococcus was recovered from the blood stream. It will be noted that a staphylococcus was the causative organism in cases 2 and 4 of the present series.

Complications may be considered as immediate or remote. Immediate complications are those due to direct pressure or extension, and include such morbid processes as bone necrosis, meningitis in all its various forms, brain abscesses, extension of the infective process into communicating sinuses, and neuritis of any or all of the intracranial nerves. The remote complications are the result of the accompanying bacteremia with resulting metastatic abscesses of the brain, lungs, muscles, joints, spleen and kidneys. Toxins elaborated at the primary focus of infection and accentuated by toxins from secondary foci, if such be present, are not without significance in the production of the symptom complex of sinus thrombosis, and account in part for the cachexia so frequently observed in these cases. Endocarditis is not infrequently a serious complication, and nephritis of variable degree is usually present at some time during the course of the disease.

The typical symptoms of sinus thrombosis are classical, have been described by many authors, and may be referred to in any of the standard texts. Suffice it to say that there is usually a history of a preceding purulent otitis or an acute

exacerbation of a chronic process. The onset is usually ushered in with a chill or a sensation of chilliness, accompanied by a sharp rise of temperature to 103, 104 or 105 degrees F., or more, which quickly recedes to normal or below in uncomplicated cases. The rigor, with its sudden characteristic rise and fall of temperature, is frequently followed by profuse sweating, after which there is a varying period of euphoria until the next cyclic shower of bacteria is thrown into the blood stream. The preceding symptoms are repeated in modified cycles of varying intervals. A blood culture at the time or just after the temperature has attained its maximum will usually, though not invariably, show a blood stream infection present.

A differential white cell count will reveal an increased leucocytosis with a relatively high percentage of polymorphonuclear cells present, unless there is a lack of resistance on the part of the patient, when we may encounter slight, if any, increase above the normal. The patient may complain of a diffuse or localized pain in the head, a stiff neck due to an involvement of the lymphatics along the corresponding jugular vein, with hoarseness, aphonia or dyspnea, due to pressure on the spinal accessory, glossopharyngeal and pneumogastric nerves as they emerge at the base of the skull through the middle compartment of the jugular foramen. Edema over the mastoid emissary vein, with pain on pressure, is suggestive of a thrombus of that vessel with a possible extension to the lateral sinus. Although a hard, tender, cordlike swelling along the course of the internal jugular is suggestive of a thrombus of that vessel, it cannot be relied upon, as inflamed lymphatic glands in this region may give similar findings. According to S. MacEwan Smith,[7] the development of an abscess beneath the deep fascia in the posterior cervical triangle is secondary to, and we would assume diagnostic of, infection of the posterior condylar veins.

The diagnosis of sinus thrombosis is determined by carefully weighing the symptoms and findings of the particular case under consideration. The surgeon will take into consideration the age of the patient, as this is not a disease of the extremes of life, but usually occurs between five and forty. It has its characteristic temperature curve with corresponding

circulatory and respiratory chartings. The finding of a positive blood stream infection with the foregoing symptoms is almost, if not absolutely, pathognomonic of sinus thrombosis, especially when accompanied by an increased white cell count with a high percentage of polymorphonuclear leucocytes. A differential diagnosis must be made between sinus thrombosis and other intracranial lesions, such as brain abscess, tuberculous or purulent meningitis (especially the former in children), typhoid fever, malaria, erysipelas with retarded eruption and malignant endocarditis. The difficulties of a differential diagnosis are further enhanced by the necessity of determining which side is involved, after making a diagnosis of sinus thrombosis, in a case of bilateral disease of the middle ear and mastoid cells.

A modification of the summary laid down by Campbell[4] will be of value in determining which sinus is involved:

(1) Anatomically, the right sinus is so situated in relation to the mastoid cells as to render it more liable to infection than the left in the proportion of 3 to 2.

(2) The time of incidence of the original infection has some weight, the older the process the more likely the complication.

(3) The X-ray findings on a stereoscopic plate may show a variation in type of anatomic development of the two sides, the extent of mastoid disintegration, the position of the lateral sinus and at times a shadow of the enclosed clot.

(4) Comparison of tenderness and glandular involvement below the tip and along the jugular on the two sides of the neck may be of diagnostic value in some cases, but is not to be relied upon in children.

(5) Compression of the jugular vein on the opposite side causing increased fullness of the retinal veins as determined by the ophthalmoscope is theoretically sound but difficult of execution and is of value only when the sinus is completely occluded. Although we have not had experience with it, the same objections might be raised against the manometer reading of the spinal fluid, after the manner of Toby, which is carried out by attaching a manometer to a spinal puncture needle and noting the pressure reading before and after compression of either internal jugular. Compression of an occluded jugular will not increase the manometer reading, while

compression of the patent jugular whose fellow is occluded will accentuate the reading.

(6) The character and extent of the infection and the bone destruction at the time of the primary double mastoid operation.

(7) Examination of each vessel wall after exposing both lateral sinuses from above the knee to the region of the bulb, noting the physical characteristics, and especially whether they fill upon release of pressure. Failing to obtain the desired information, we may resort to aspirating the contents of either sinus and compare the results. If necessary, additional information may be obtained by determining the bacterial content of either sinus respectively.

The treatment of lateral sinus thrombosis should be preventive, by making an early diagnosis of otitis media and instituting such general treatment as will increase the patient's resistance, combined with early paracentesis, if necessary, and complete exenteration of the mastoid cells when indicated. This, in our opinion, would prevent most cases of sinus thrombosis caused by direct extension of infection and greatly reduce the number of those resulting from metastasis.

Once the thrombus is established, the consensus of opinion among otologists is for surgical interference. Ligation of the internal jugular vein, with or without resection, is probably favored by most otologists. There are those who, on exposing the sinus and opening the same, obtaining free bleeding, reserve the ligation or resection until such future time as circumstances demand more radical measures.

Koebbe[8] reports bringing the upper or proximal end of the nonbleeding internal jugular vein into the wound without ligating for drainage. We have observed one case in which bleeding could not be induced from the lower end of the sinus, or from the upper end of the internal jugular, in which a normal saline solution was gently forced by gravity through the unligated severed internal jugular vein and made to flow into the mastoid wound from the lower end of the sinus. There are some few operators who always ligate the jugular before exposing and exploring the sinus. Regarding the latter, we can only say that there is no positive indication for ligation of the internal jugular vein until the mastoid has been explored,

because by this means only can the operator determine the exact condition of the sinus and its contents.

Blood transfusions have become a recognized procedure in the treatment of sinus thrombosis and are not only of great value in those cases of bacteremia with a doubtful thrombosis but are bactericidal and supportive in cases of obliteration of the lateral sinus and in cases of ligation and resection of the internal jugular vein following a mastoidectomy in which intermittent temperature persists.

Case 1 (S. 527).—H. S., male, aged thirteen years, came to the office March 22, 1922, in such intense pain that he repeatedly requested to be hit on the head with an ax. Previous and family histories were negative as to the present complaint, which was that he had been ill for three weeks, the last two of which he had been confined to his bed with a fever and an unbearable pain in the left ear and corresponding side of the head. Left ear "broke" three days ago. The temperature had not been taken, as he had not received medical attention.

The patient gave the appearance of being ill and distressed. He was pale and anemic and had an anxious look. It was noted that the left ear bulged forward and downward away from the head. There was a marked swelling over the zygomatic area on the corresponding side.

An examination disclosed the right ear normal; the left, as above noted, with the canal filled with purulent secretion and the superior portion of the canal sagging. The area over the mastoid was swollen, inflamed and exceedingly tender to pressure, as was the zygomatic region. The temperature was 103° F. White cell count showed total leucocyte count 15,000; polymorphonuclear, 85 per cent. The X-ray, as reported by Dr. C. H. Thompson, was, "Cells in right mastoid clearly shown. Those in left blurred and obscured. Swelling over zygoma not shown in radiograph."

The following morning a complete mastoidectomy was performed. The cellular area was extensive and there was a marked destruction of the bone, especially in the posterior root of the zygoma, in which region the dura was exposed and a large extradural abscess was evacuated and drained. The sinus plate was intact and not molested. Recovery was uneventful.

After having gone through such an ordeal, and after having

been duly impressed with the fact that such maladies brook no delay, it would be expected that such an experience would have been taken to heart and long remembered. Such, however, was not the case with this patient and those responsible for his welfare, for on April 1, 1924, we were again consulted for the following complaint and symptoms: It was stated that his general condition had been excellent since the operation two years ago. There had been no discharge from the ear and he had been free from pain and headaches until four weeks ago, when he suffered from an attack of acute follicular tonsillitis. This was soon followed by an intense pain in the ear and by a purulent discharge from the canal on the side previously operated on. There was marked swelling over the mastoid area, which was painful and tender to the touch, and there was a heavy, copious, mucopurulent discharge from the canal. Temperature was 102.2° F., pulse 124. Urine negative except for a slight trace of albumin.

The patient was admitted to the hospital, and on the following morning, April 2nd, under ethyl chlorid and ether anesthesia, the scar of the previous operation was excised and pus evacuated. A fistulous tract leading into the region of the previous extradural abscess was discovered, widely opened and the area drained. A culture taken at this time proved the infection to be due to a short chained streptococcus. The upper angle of the incision was closed with silkworm gut suture, after the wound had been lightly packed with iodoform gauze. Dressings were applied and the patient returned to the room in good condition. The postoperative convalescence was satisfactory, except the sutures were under tension and were removed on the second day, leaving a widely gaping wound.

Because of an unavoidable absence from the city, it became necessary to relinquish the care of the patient to another surgeon who, deeming the patient's condition, both general and local, as favorable, unwittingly discharged him from the hospital after a sudden rise of temperature (102° F.).

We next saw the patient on May 8th, at our office, just one month after he had been discharged from the hospital. Although he was up and about, his condition at that time is more easily imagined than described. He was so weak from chills, hyperpyrexia and sweats that he could scarcely get about. He

was emaciated. His eyes were sunken and his skin cold and clammy. His appearance was spectral, to say the least.

An X-ray report by Dr. H. B. Thompson described an area of large pneumatic cells in the right mastoid, the cells being of the mixed variety. The pneumatic area extended well forward and above the auditory canal, as well as posterior. On the left side the cells above the auditory canal had apparently been removed, while those posterior to the canal could still be faintly made out, although apparently filled with material. Above the auditory canal there was a small circumscribed area of increased radiability. It was impossible to determine whether this was due to operation or bone softening from disease.

He was admitted to the hospital two days later with a temperature of 104.6° F. and with a pulse of 160. Urinalysis negative. Leucocyte count, 10,000; polymorphonuclear cells, 74 per cent. Two consecutive blood cultures were negative, 24 and 48 hours after entrance.

Although the eyegrounds were negative and there was an absence of neurologic symptoms, the patient was sent to the surgery with a diagnosis of left sinus thrombosis, osteomyelitis of the temporal bone and possibly an extradural abscess.

Under gas ether anesthesia, the left internal jugular vein was exposed and ligated by Dr. Conrad Jacobson, who made an incision along the anterior border of the sternocleidomastoid muscle. A cigarette drain was inserted and the upper portion of the wound closed. He then made a scalp incision two and one-half inches in length, two finger breadths above the auricle, and trephined the temporal bone. As exposure and exploration of the dura and brain in the temporosphenoid lobe area revealed nothing to account for the clinical symptoms, the scalp wound was closed.

The mastoid wound was reopened and all granulation tissue curetted away, exposing both the tegmen tympani, which was removed, and the sinus plate, which was intact and of normal consistency. The removal of this shell of bone revealed a discolored, doughy vessel with thickened walls. Incision of the vessel wall released thick, creamy pus, which under the microscope showed pus cells and an occasional diplococcus which failed to grow on culture.

As these operative procedures were time consuming, and the condition of the patient none too good, it was deemed best not to break down the natural barriers and attempt to obtain a flow of blood from either end of the sinus but to treat the case as a localized abscess, and, to that end, a strip of iodoform gauze was inserted directly into the sinus, both above and below, to act as a drain. A drain was also inserted under the mastoid wound up to and into the region of the trephine opening. Another drain was inserted into the antrum, and dressings applied without closing the mastoid wound.

The patient made a slow but uneventful recovery, and when last seen gave no evidence of his varied experiences, except the inevitable scarring, which was of little consequence.

Case 2 (W. 543).—B. W., female, aged ten years, was admitted to the hospital March 7, 1924, with a purulent discharge from the right ear and slight tenderness over the corresponding mastoid region. There was induration and swelling over the internal jugular on the right side.

Family history was negative. The present history, as given, was that the tonsils and adenoids were removed two months ago. The tonsil operation was followed by a discharging right ear. Because of poor general condition, the child was admitted to an institution, where she contracted measles three weeks ago. The ear symptoms were accentuated, and on March 5th, while en route to consult a pediatrician regarding the same, she vomited while on the street car and again while in his office. The temperature at that time was 105° F. That evening it rose to 106° F., and on March 9th, it reached the high mark of 107.6° F.

The physical examination at the time of admission showed a well developed and well nourished child, with normal eyes, nose and throat. The heart, lungs, abdomen and extremities were normal. The reflexes were hyperactive. There was a profuse discharge from the right ear, with slight tenderness over the mastoid area, especially over the tip. The neck was rigid and tender over the jugular, below the tip, on the right side. Temperature at that time was 101° F. Leucocyte count 10,500, with polymorphonuclear cells 74 per cent. Blood culture showed a growth of staphylococcus after 24 hours. The child was listless and appeared extremely ill.

The X-ray findings, by Dr. H. B. Thompson, indicated the left mastoid area of good size, the cells being of the small variety. The right mastoid showed a definite clouding, with apparent destruction of the cell walls in the posterior portion, pointing to the presence of pus or granulations, with probably some bone destruction.

March 10th, a complete simple mastoidectomy was performed on the right side, during which it was noted that there was a moderate amount of pus and granulation tissue present and many of the cells broken down. The meninges in the region of the tegmen antri were unintentionally uncovered and found to be normal. The lateral sinus was exposed in the region of the knee for an extent of three-fourths of an inch, and evidence of thrombosis was present.

Because of the sinus findings and the previous clinical course, the neck was prepared and an incision along the anterior border of the sternocleidomastoid muscle made. The internal jugular was isolated and found collapsed. The vessel was severed above the facial, between ligatures of chromic gut, and the upper portion of the wound closed, with a drain inserted.

The sinus was opened and there was free hemorrhage from the upper end immediately, but not from the lower end until a thrombus was dislodged. The bleeding was controlled by the previous insertion of small rolls of iodoform gauze wedged in between the sinus and bony wall of the skull. The mastoid wound was packed lightly and the upper portion closed as usual.

Dressings were applied and the patient returned to the room with only a moderate amount of shock, for which caffein was ordered, and after three-fourths of an hour 300 cc. of normal saline solution were given intravenously.

The patient ran a more or less intermittent temperature, for which 300 cc. of blood were given, by the citrate method, intravenously, on the eighth postoperative day. Following this there was an uneventful recovery, except for the development of several small superficial skin abscesses, one of which required an incision.

The patient was discharged April 6th, in good condition, with both operative wounds closed and the ear dry.

Case 3 (B. 772).—M. B., aged ten years, was first seen March 2nd, 1924, at which time the following history was elicited: Measles two months ago. Never had a discharge from or a pain in either ear before the present attack, which followed a plunge in a local swimming pool three days previous. The day following the plunge there was an excruciating pain in the left ear, relieved by a serosanguinous discharge, through a spontaneous perforation of the tympanum. On the second day the little patient was dizzy and vomited frequently, without apparent reference to food intake.

An examination on this, the third day of the disease, showed an acute simple rhinitis, turbinates hypertrophic, adenoids and tonsils removed, with a mucopurulent discharge in the nasopharynx. Nasal sinuses negative to inspection. The right ear was normal. A bloody, serous discharge was flowing from the left ear. No tenderness over the mastoid tip or antrum. The child was bright and comfortable, with a temperature of less than 100° F.

Two days later I was called and found the child dull and apathetic, with a temperature of 102° F. The discharge from the left ear was the same as before. The right ear had ruptured twenty-four hours previously, and the external canal was filled with a purulent discharge. There was tenderness over the left mastoid antrum and tip, and pain extending down the back of the neck. There was a sagging of the posterosuperior portion of the canal of the left ear. Landmarks of the tympanum were not to be made out. Temperature 100° F., facial expression anxious, and, as there was intermittent vomiting, the patient was admitted to the hospital for surgical intervention.

The routine hospital examination, as recorded, showed total leucocyte count 15,800; the percentage of polymorphonuclear cells was 84 per cent and the urinalysis was negative. The X-ray report (by Dr. J. T. Dawson) stated that, "With the exception of an abscess of the anterior cells (on the left side), we find no acute pathology in either mastoid."

A simple mastoidectomy was performed under ether anesthesia on the left side March 5th. Upon removing the cortex an extensive small celled type of bone, with large cells at the tip and over the knee of the sinus, was noted, with the sinus plate

well forward. The infectious process was of the thrombophlebitic type, and moderate bleeding was encountered throughout the course of the operation. The sinus plate, which was intact and of a bluish color, was extensively removed, disclosing an organized blood clot extending over the lower half of the exposed sinus area down to the jugular bulb. The clot was stripped from the vessel wall, and blood, which appeared normal, was aspirated from the lumen of the same by the insertion of a small needle attached to a 10 cc. Luer syringe. The cells, including those at the angle of the knee, tip and gutter region, were removed and the antrum widely opened. The plate of the middle fossa was found intact and not molested. The upper portion of the wound was closed, with a drain inserted to the antrum. Dressings were applied and patient returned to the room in good condition.

The postoperative convalescence was uneventful until June 11th, when the patient complained of pain in the right elbow, which was found to be swollen and inflamed. Upon immobilization of the joint and the application of heat this complication subsided. The right ear ceased to discharge, and on the 18th of June the patient was permitted to leave the hospital in good condition, although there was some unhealthy granulation tissue in the mastoid wound.

The patient was cared for and the wound dressed at the home for nearly a week, after which time chills, vomiting and pain in head occurred, with an intermittent temperature of 98 to 104 degrees F.

The patient was readmitted to the hospital June 26th, with a diagnosis of left lateral sinus thrombosis. Leucocyte count 16,200, polymorphonuclear 81 per cent. Two blood cultures were negative after twenty-four hours. Urinalysis disclosed hyaline casts and trace of albumin.

On June 28th, under ether anesthesia, the left internal jugular vein was exposed and severed between ligatures. A cigarette drain was inserted into the wound at the lower angle, after which it was closed with interrupted silkworm gut sutures. The mastoid wound was reopened, and the recently formed granulation tissue removed. The exposed and thickened sinus wall was incised longitudinally, after having first inserted a small pledget of rolled iodoform gauze between the

inner table and the sinus wall at either end of the exposed vessel. Bleeding was free, both from above and below. Here our trouble began, as the rolls of iodoform gauze did not control the hemorrhage, and we were compelled to reinforce them with a gauze pack directly over the sinus. The wound was left wide open, dressed and bandages applied.

The patient left the operating room in fair condition, in spite of the loss of blood, which had raised the pulse to 170 and increased the respirations to 36 per minute.

The patient's condition was satisfactory during the next few days, except for moderate nausea and vomiting. On July 2nd, under ether anesthesia, the pack was removed from over the sinus area, and on July 5th an attempt, under anesthesia, was again made to remove the pledgets of iodoform gauze. Free hemorrhage followed, necessitating reinsertion of both pledgets and the pack. July 9th, there was a complete paralysis of the left external rectus, with diplopia. Ophthalmoscopic examination disclosed a choked disc on the left and a similar process beginning on the right. On July 10th, we were able to loosen the upper pledget and dispense with the pack. It was not until July 14th, under ethyl chlorid anesthesia, that we were able to remove both pledgets and the pack, after which the child improved rapidly and was discharged July 24th, or exactly two weeks after the removal of the pack.

It is most gratifying to record that the choked discs and the paralysis of the external rectus entirely subsided after a few weeks and the child regained and has remained in excellent health.

Case 4 (B. 742).—G. B., female, aged 16 years, was first examined at the office March 8, 1924, at which time there was a history of intermittent earache in the right ear since early childhood, resulting in an impairment of hearing in the corresponding ear. The patient had measles nine years ago, chickenpox seven years ago and mumps five years ago. There had been a remission of ear symptoms for a time after a tonsillectomy six years ago, followed by a complete subsidence of the trouble until one year ago, when there was a flare-up that subsided without treatment. Six months ago slight pains occurred in the ear. The pains had been worse during the last month and had progressively increased in severity until

the preceding night they became severe, with a dull, heavy pounding in the head, on the same side, that interfered with the patient's rest. There was no evidence of discharge until yesterday, when a slight amount of secretion was found on a small pledget of cotton.

The family history was negative. By a casual examination it was noted that the patient was phlegmatic, rather large and obese for her age. Temperature normal, pulse 74. Examination of the affected ear showed a dry canal, with no evidence of discharge, no sagging of the posterosuperior portion of the canal or bulging of the drum membrane. There was a small perforation in the membrana flaccida. There was no evidence of swelling or tenderness over the mastoid area or in the cervical region. The patient was given such attention as was demanded at the time and advised to report the following day for observation.

Nothing was heard from the patient for four days, when I was called, at the request of her family physician, to see the patient in consultation. The patient was in bed with what was diagnosed as an attack of influenza.

The day following the first examination the patient went to bed with a pain in the lower midabdomen. The pain was intense and steady in character, and was accompanied by nausea and vomiting. The vomiting had since continued daily. The action of the bowels was normal the first two days, but later the movements became very loose, and there had been three or more daily evacuations since.

Examination of the ear at this time revealed the same conditions present as when first examined, with the additional history of a feeling of chilliness. As the patient's illness was considered to be more of a general than a local nature, she was permitted to pass from our observation until March 16, 1924, or four days later, when I was requested to examine her at the hospital. The following summary of her findings were recorded on the chart: "Temperature 98 to 104.8 degrees F. Pulse 96. Generalized acute abdominal tenderness. Although there is no definite localization, the left side and bladder region appear more tender on pressure than other abdominal areas. Patient denies gonorrhea, leucorrhea or burning and smarting on urination. Nervous system negative. The patient is having

chills and running a temperature curve of septicemia, the probable result of an abscess in the left lower abdominal quadrant. Repeated blood cultures are advised to rule out or confirm the diagnosis."

The examination of the right ear showed the drum injected, with the perforation as previously described, otherwise the landmarks were obscure. But little, if any, secretion present in the canal. There was slight tenderness below the tip of the mastoid and over the internal jugular vein on deep pressure. Pupils equal and normal to light and accommodation. No nystagmus detected, and the nerve heads were normal.

The left ear was negative. Advised X-ray of the mastoids for comparison and repeated blood cultures, as there was a probable thrombosis of the right lateral sinus.

The X-ray report, as given by Dr. H. B. Thompson, stated that: "In the right mastoid there is practically complete obliteration of all the cell wall markings, and a definite diminution in size of the pneumatic area, the outer portion apparently being sclerosed. The median portion of the right mastoid area shows several irregular areas of increased penetration, suggesting the probability of localized bone destruction and abscess formation. The left mastoid shows a large pneumatic area, the cells being the small celled variety." The blood culture was negative after 48 hours.

Because of the inability to make a diagnosis of the abdominal condition, it was decided, on March 18th, to exenterate the mastoid cells and explore the sinus. Under gas ether anesthesia, the mastoid bone was found to be very hard and sclerosed, with the periosteum firmly adherent to the bone. The mastoid plate, which was intact, was superficial and well forward. The mastoid process contained but few cells, which were removed with difficulty, because of the excessive sclerosis. The sinus plate was then removed over the widest possible area, and a doughy sinus, with thickened walls, was exposed.

Dr. C. B. Ford exposed and ligated a collapsed internal jugular vein with thickened walls, by the usual incision along the anterior border of the sternocleidomastoid muscle. A small rubber drain was inserted to the bottom of the wound and its upper angle closed with catgut and dermal suture.

Small rolls of iodoform gauze were then inserted, both above and below, between the sinus wall and the inner plate of the skull, to control the hemorrhage. The sinus wall was then incised and free bleeding established from both above and below. The rolls of iodoform gauze were reinserted and the mastoid wound closed in its upper three-fourths with a gauze pack loosely inserted.

The convalescence, although stormy and protracted, was very gratifying. It is convenient and proper at this time to report that, some three or four days after the mastoid operation, the nurse in attendance reported the presence of a profuse purulent discharge from the vagina, which, to the mind of the writer, was the result of a ruptured abdominal abscess secondary to the thrombosed lateral sinus, and was the cause of all the abdominal symptoms.

If the above deductions are correct, then it is most fortunate that Nature gave timely aid to this patient by evacuating the contents of the abscess in the manner above described.

Case 5 (H. 711).—J. H., male, aged 6 years, was first seen in consultation at his home, April 15, 1925, at which time it was ascertained that three days previously he had had a temperature of 104° F., and had taken to his bed with an acute tonsillitis, which was soon complicated by pain in the left ear. The drum ruptured spontaneously within a few hours, giving marked relief. The temperature had continued high, and the child had vomited several times. At the time of the first examination, I found a bright, well nourished child, whose family and personal histories were negative. The right ear was normal. The left showed a small perforation in the posterior inferior quadrant, after I had removed a small amount of mucopurulent secretion from the canal. The drum was injected and the normal landmarks were obscured. There was slight tenderness over the mastoid antrum and tip. The cervical glands were enlarged on either side, especially the left. There was a moderate acute rhinitis and tonsillitis. The child had a well defined punctate rash, of only a few hours' duration, peculiar to scarlet fever. Appropriate treatment was advised and prescribed, and the case left in charge of the attending pediatrician, who was warned of the possibilities of complications.

On being called in four days later, the little patient was

found with a temperature of 104° F., the posterosuperior portion of the canal sagging and a marked swelling below the tip of the mastoid, extending well into the cervical region. The mastoid area, especially the tip, was tender on pressure. The child was admitted to the scarlet fever ward at Firland's Sanitarium and the next morning a simple mastoidectomy was done.

During the operation, which was performed on April 20, 1925, under ether anesthesia, a mastoid of the small celled variety was exenterated, leaving the sinus plate, the tegmen tympani, unmolested and intact. The antrum was widely opened and drained. The cells in the tip were removed. The upper portion of the wound was closed with interrupted silkworm gut sutures and dressings applied. The patient was taken to his room in good condition.

The following day the child complained of pain in the right elbow and would make no attempt to use it. The elbow was immobilized and general supportive treatment given. A blood count at this time gave 10,500 leucocytes, with a polymorphonuclear count of 84 per cent. The temperature was 100.2° F. The child continued to run an intermittent temperature of 100 to 104 degrees F., and the pain, which had been previously localized in the elbow, was referred to the right shoulder, which became red, swollen and painful. An X-ray picture of the acromiclavicular joint was of doubtful diagnostic value.

The intermittent temperature continued, and the child seemed to be losing ground. This we knew was due to the focus in the shoulder, a sinus thrombosis or the effect of a vaccination against smallpox, on admission to the hospital. On April 27th the white count was: Leucocytes 19,500, with a polymorphonuclear percentage of 84. We regret that the lack of facilities precluded the taking of blood cultures. The patient continued to run an intermittent temperature without any more evidence of rigors than possibly a slight chilliness. The fundi were normal on examination.

On April 30th, or ten days after the primary mastoid operation, the child was taken to the operating room and, under ether anesthesia, Dr. R. L. Jeffery explored and evacuated pus from the shoulder and instituted Carrel-Dakin treatment. While under the anesthetic the sinus plate was removed, disclosing a

dark doughy sinus with thickened walls. A pledget of gauze was inserted between the sinus and the bony wall of the skull, both above and below. The sinus wall was incised longitudinally and free hemorrhage occurred from either end of the sinus. The sinus was obliterated by the reinsertion of the gauze rolls and drainage facilitated by leaving the mastoid wound open. Dressings were applied to both wounds and the child returned to his room in good condition. The Carrel-Dakin solution was continued until the wound at the shoulder had cleared and ceased to drain. Both pledgets of gauze were removed on the fifth day, and thereafter the wound was dressed as any simple mastoid.

The child, except for whooping cough, made a slow but uneventful recovery and was discharged from the hospital as soon as quarantine regulations would permit. His condition at this time was excellent, with the shoulder wound closed and the mastoid wound filling in as rapidly as could be expected under the circumstances.

Twenty slides were shown, illustrating the preceding cases.

CONCLUSIONS.

1. Any case of mastoid infection may be complicated by lateral sinus thrombosis.

2. In a large series of mastoid infections the incidence of thrombosis runs between 3 and 6 per cent.

3. In suspected cases of sinus thrombosis repeated daily blood cultures should be taken, and at the same time a differential white count should be made; also, the temperature readings should be graphically charted every two hours.

4. The lateral sinus should be uncovered and inspected in all operations for mastoiditis suspected of being complicated by a thrombus.

5. In the majority of cases in which, on exploration of the sinus, free bleeding is obtained from above and below, the surgeon is justified in postponing ligation or resection of the internal jugular for further consideration.

6. If free bleeding from the lower end of the sinus is not obtained, then inspection of the internal jugular, with ligation or resection, as indicated by sound surgical judgment, is in order.

7. Once the the diagnosis of sinus thrombosis is established, the sooner properly directed surgical measures are instituted the less will be the operative mortality and the fewer the complications.

8. Each case is a subject for individual study, and the question whether to operate or not can only be determined by the operator's experience and surgical judgment.

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LI.

A CASE SUGGESTIVE OF EMBOLUS OF A COCHLEAR ARTERY.*

By Isaac H. Jones, M. D.,

Los Angeles.

Patient: Mr. G. A. L., age 50. Examined September 13, 1925.

History: He stated that on January 29, 1925, he went to bed with excellent hearing in both ears and woke up the next morning deaf in the left ear. His only complaint was a severe cold in the head; discomfort because of a sense of great pressure between his eyes and inability to breathe through his nose; no headache, no earache, no sore throat, no cough. He had taken no medicine. He went promptly to sleep in spite of the cold in the head and slept well through the entire night without any pain, vertigo, nausea or any other symptom. When he awoke he noted that he could not hear the watch in his left ear and that the left side of his head felt dull, and that there was a roaring "like a seashell" in the left ear. He was treated with astringents, catheter and Politzeration without relief. On March 10th he consulted Dr. Donoher of Salt Lake City, who noted that the middle ears appeared normal and showed normal movement with pneumatic otoscope; that the tinnitus consisted not only of the sound of the seashell but another sound which came in very suddenly, at almost hourly intervals and lasted about thirty seconds. This peculiar sound could be described perfectly by the tapping of an "A440" fork with a lead pencil. Blood Wassermann was reported negative. Dr. Donoher made a diagnosis of involvement of the left eighth nerve, of unknown origin.

Family: Father died at 52 of pleurisy; mother at 55 of cancer of the uterus; one brother, at 16, of tubercular hip, following an accident; another brother, at 17, was stricken

*Read before the Western Section of the American Laryngological, Rhinological and Otological Society, Los Angeles, Cal., January 29, 1926.

with intense pain in the head, lost consciousness three hours later, and died in three days—presumably meningitis; a sister died at the age of two of meningitis after being sick five days.

Previous: Has had excellent health and vigor all his life. No accidents or operations. States he has occasional mild attacks of sore throat, but has paid no attention to them.

Examination in Los Angeles, September 13, 1925, showed chronic catarrhal changes in accessory sinuses; no active secretion. Oral mucosa mottled. Teeth in fair condition; none capped. Palate negative. Faucial tonsils deeply buried and red. Definite chronic catarrhal pharyngitis. One large gland on either side of anterior cervical chain, and tenderness along the anterior lymphatic chain, even though the glands of the chain are not enlarged. Drum membranes negative, except slight retraction of the left. Slight but definite vestibular subnormality is shown by the caloric tests. Audioamplifier shows good cochlear function in right ear and very little cochlear function in the left ear. The details are given on the accompanying vestibular chart and audioamplifier chart.

Comment: To the limit of my personal knowledge, this is an unusual condition—sudden unilateral deafness without any vestibular involvement. It is obvious that there was no vestibular involvement because there was a complete absence of any vestibular symptoms at the time of the attack, and also because the vestibular examination shows a fairly good nystagmus after douching the left ear for forty-five seconds. The lesion, therefore, would seem to be either in the cochlear branch of the left eighth nerve or in the left cochlea itself. A sudden neuritis of the left eighth nerve, which would so markedly impair the function of the cochlear portion, without affecting its vestibular portion, would seem improbable. It seemed to me that the most reasonable diagnosis is that an embolus, from the tonsils perhaps, had lodged in a terminal branch of an artery supplying the left internal ear. Shambaugh states that approximately two-thirds of the basal coil of the cochlea is supplied by a branch of the labyrinthine artery; however, this same branch also supplies the macula of the saccule and the crista of the posterior semicircular canal. The branch of the labyrinthine artery which runs through the modiolus and supplies all of the cochlea, except the proximal two-thirds of the

basal coil, sends off a branch which goes to the macula of the utricle and the cristæ of the horizontal and the superior semicircular canals. In brief, it has seemed to me that an embolus interfering with the blood supply to the cochlea would best explain the findings in this case; however, such a diagnosis would not explain why there was no disturbance along the distribution of the arterioles to the maculæ and cristæ.

From the date of onset to the present day, a period of one year, there has been no change in the condition of the patient. He is in perfect general health and still remains deaf in the left ear.

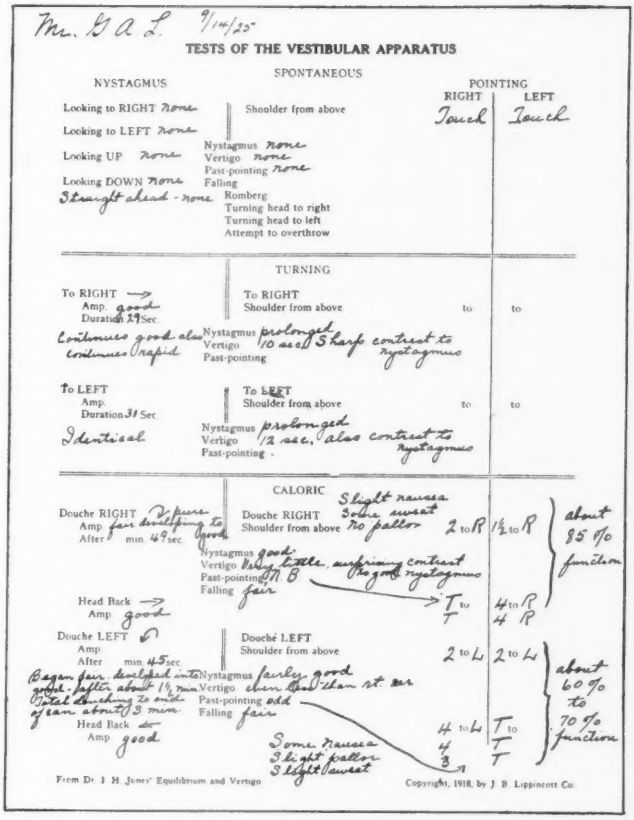

Mr. G A L 9/14/25
TESTS OF THE VESTIBULAR APPARATUS
NYSTAGMUS
SPONTANEOUS
POINTING
RIGHT
LEFT
Looking to RIGHT none
Looking to LEFT none
Looking UP none
Looking DOWN none
Straight ahead - none
Shoulder from above
Touch
Touch
Nystagmus none
Vertigo none
Past-pointing none
Falling
Romberg
Turning head to right
Turning head to left
Attempt to overthrow
TURNING
To RIGHT →
Amp. good
Duration 29 Sec
To RIGHT
Shoulder from above
to
to
Continues good also continues rapid
Nystagmus prolonged
Vertigo 10 sec
Past-pointing
Sharp contrast to nystagmus
To LEFT
Amp.
Duration 31 Sec
To LEFT
Shoulder from above
to
to
Identical
Nystagmus prolonged
Vertigo 1/2 sec.
Past-pointing
Also contrast to nystagmus
CALORIC
Douche RIGHT
Amp. fair developing to good
After min. 49 sec.
Douche RIGHT
Shoulder from above
Slight nausea
Some sweat
No pallor
2 to R
1/2 to R
about 85 % function
Nystagmus good
Vertigo Very little, suffering contract No good nystagmus
Past-pointing M B
Falling fair
T to T
4 to R
4 R
Head Back →
Amp. good
Douche LEFT ↶
Amp.
After min. 45 sec.
Douche LEFT
Shoulder from above
2 to L
2 to L
about 40 % to 70 % function
Began fair developed into Vertigo, better about 1/2 min. Vital reaction to cold of ear about 3 min.
Nystagmus fairly good
Vertigo about as at ear
Past-pointing odd
Falling fair
4 to L
T to T
Head Back ↶
Amp. good
Some nausea
Slight pallor
Slight sweat
4
3
From Dr. J. H. Jones' Equilibrium and Vertigo
Copyright, 1918, by J. B. Lippincott Co.

Chart 1.

AUDITORY CHART.

Name Mr. G.A.L. Date 9/13/25

How old when hearing impairment first observed? January 1925

Describe onset. Got up one morning, deaf left ear

Hear better in presence of noise or in quiet room? Quiet

Any deafness in family? No

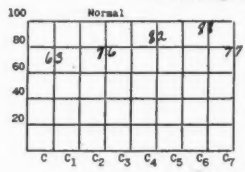
How hear over telephone? Not with left ear, radio? Not tried

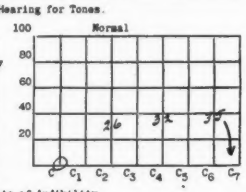
Tinnitus? Slight seashell roaring. Occasional clicking as if air were getting through.

Measurements with Audio Amplifier.

RIGHT EAR.	I. Amplifier.	LEFT EAR.
Voice, confused ____, distinct Fair		Voice, confused ____, distinct ____
Heard best with uniform amp ____		Heard best with uniform amp ____
Low pass amp ____, High pass amp ____		Low pass amp ____, High pass amp ____
Whisper, percentage hearing 100%		Whisper, percentage hearing None

II. Percentage Hearing for Tones.





III. Upper Limits of Audibility.

17000 d.v. 8500 d.v.

IV. Bone Conduction.

128 ____, 256 93%, 512 79% 128 ____, 256 86%, 512 83%.

V. Special Findings.

(Pitch and loudness of tinnitus, diplacusis, tonal islands, etc.)

Diagnosis: Right cochlea good function.
Left cochlea marked defect

Comments: ____

Note: C – 64 d.v., C_1 – 128 d.v., C_2 – 256 d.v., C_3 – 512 d.v., C_4 – 1024 d.v., C_5 – 2048 d.v., C_6 – 4096 d.v., C_7 – 8192 d.v.

Chart 2.

LII.

A PLAN FOR GROUP TESTING OF THE HEARING OF SCHOOL CHILDREN.

By Vern O. Knudsen, Ph. D.,

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AND

Isaac H. Jones, M. D.,

Los Angeles.

Ordinarily it is not good taste to present an outline of experimental work before the work actually has been performed. However, in the present paper this procedure has been chosen deliberately. If group testing of the hearing of school children be undertaken here, or elsewhere, it has seemed to us that many regrets could be avoided if the plan first be discussed by those who are in a position to advise. To be of any statistical value, for example, it is necessary that the final charts and records should be the same as those which are used from the beginning. Before any work of this kind is actually done, we should know most definitely and completely just what procedure will be the most fruitful and practical. It is with this purpose in mind that the following tentative plan is presented at this meeting:

We all know, to our regret, how little we can do to "restore the hearing to the deaf." In fact, there is probably no more unsatisfactory phase of ear, nose and throat work. When we make the most exhaustive study of a patient; determine the type of deafness; make the most precise measurements as to the degree of deafness; and, in many instances, determine even the precise cause of deafness—it is only to inform the patient that "we may be able to do something to arrest the progress of the condition." It is not our wish to belittle the many efforts that are being made to treat the various types of deafness. However, we feel that the one real opportunity for us

to be of service lies very largely in prevention. Now that instruments of precision are available whereby a beginning deafness may be detected five, ten or fifteen years before it becomes noticeable to a child, its parents or its teachers, it has occurred to several investigators, who are familiar with these instruments, that a new opportunity arises in approaching the problems of deafness. From the idealistic standpoint, it would seem advisable to make precise measurements of all the school children in the world; to detect a pathologic process before it has produced permanent or more or less irreparable changes in the hearing mechanism, and then to have suitable treatment instituted during these early stages. It does not appear incredible, if such a dream could be realized, that a very appreciable percentage of deafness could be prevented.

From the strictly practical standpoint, we feel that in recent instruments of precision we have a potent means for group, as well as individual, tests of hearing. Any hearing test made, for example, with tuning forks requires the skilled otologist. Further, with the tuning forks it is not possible to examine more than one individual at a time. On the contrary, with the recently developed audiometric instruments it is a fairly simple matter to instruct any intelligent person to make tests that are not only far more precise than those made by the skilled otologist with the tuning forks, but it is also possible to test a group of individuals at the same time.

Different plans have recently been suggested for such a study. Dr. Harvey Fletcher of the Bell Telephone Laboratories has recommended the phonograph audiometer, which consists of a high quality phonograph connected with an amplifier and a volume control. As many as twenty individuals can listen at the same time through individual telephone receivers. The phonograph reproduces spoken numbers at periodic intervals, and the loudness of these reproduced spoken numbers is diminished, by means of a volume control, to inaudibility. The individuals write down what they hear until ultimately they begin to make many errors and then cease to hear. By checking up their written charts it is possible to determine the approximate threshold of hearing, from which the percentage hearing is then estimated. This test determines which individuals are

hard of hearing, and these are segregated for further, more careful, examination.

Another method, which we present for your criticism, may be described briefly as follows: A group of at least sixteen can be tested for low, medium and high tones. Those who show less than 90 per cent of normal hearing, for any part of the tonal range, are segregated, and at a later time given a more thorough examination. Similarly, at least two other groups of sixteen each are tested. Then those with defective hearing are tested for the amplified voice, the amplified whisper and bone conduction. In this manner we could obtain a rather complete report for each child who had even a slight hearing defect.

An arrangement of apparatus for testing a group of sixteen is shown in Fig. 1. In the figure, A. A. is an audioamplifier,* which develops sufficient energy for sixteen receivers. The sixteen receivers are distributed among the sixteen seats so that there is a receiver for each individual. A tone is produced in the telephone receivers and each individual signals the operator if he hears the tone. This is done by pressing on a push button at his desk, which is connected with a signal lamp, in plain view, directly under the eye of the operator. The sixteen signal lamps are numbered from one to sixteen, corresponding to the sixteen individuals being tested. Each individual puts the receiver to his right ear, and as long as he hears any tone in the receiver he keeps the button pressed. When he ceases to hear it he releases the button. The operator records a reading for each individual as he notices the light go out. Then this procedure is repeated for the left ear. The first measurement made is for the upper limit of audibility. The next measurements consist of determining the minimal thresholds for tones of 4096 d. v., 1024 d. v., 256 d. v. and 64 d. v. From these minimal threshold readings the percentage hearing for each tone is read off from a chart. On first thought this would appear to be laborious, but in a group of sixteen there probably would not be more than one or two who would not have normal hearing. The only readings, therefore, that would have to be taken would be those for the one, or ones, with the subnormal

*The Western Electric audiometer, with certain modifications, would serve equally well.

hearing. On the average, we should not expect more than one hard of hearing person in a group of sixteen. No data are recorded for those who have more than 90 per cent of hearing. Those who show more than 90 per cent of hearing for all tones, and have normal upper pitch limits, are promptly dismissed.

An examination of such a group of sixteen occupies about ten minutes. The general plan would be to examine two or three groups of sixteen in the first half hour and to spend another half hour on the more careful examination of those with hearing defects.

The data for a group of sixteen may be conveniently recorded on such a chart as is shown in Fig. 2. This provides for a record of the grade, the school, the name, the number assigned to each individual, the age, the sex, the percentages of hearing and the upper pitch limits for both right and left ears. In this sample sheet it is noted that all except No. 13 and No. 16 have no data recorded opposite their names. This signifies that all of the sixteen, except these two, had more than 90 per cent of normal hearing for all tones. No. 13 suggests evidence of a conductive hearing defect, and No. 16 suggests evidence of a perceptive hearing defect. No. 13 and No. 16, therefore, are segregated for further tests, and the others are all dismissed.

We will presume that three groups of sixteen have been examined and that five have been found to have hearing defects. In the second half of the hour these five are examined for amplified conversation, amplified whisper and bone conduction. Individual tests are made on these five individuals and recorded on a chart similar to that shown in Fig. 3.

If forty-eight are examined every hour, and if the examinations are conducted for periods totaling six hours a day, it would be possible, with only one instrument and one operator, to examine 288 individuals a day, 1,290 in a week and 46,260 in a school year. This would appear to be a goodly number until we stop to consider that in the public schools of Los Angeles alone there are 170,116 school children. However, with four sets of apparatus and four operators we would be able to conduct tests upon all of the school children in the public school system of Los Angeles in one year. In some

such manner as this, it is not beyond reason to anticipate that a hearing defect would be not only detected but also would be accurately measured and classified into its type of deafness, probably in 3,000 to 6,000 children.

From the standpoint of statistics, an investigation of the type here described would give answers to the following questions: What percentage of such a large group of school children have more than 90 per cent of hearing? What percentage have from 80 to 90 per cent hearing? From 70 to 80 per cent hearing? From 60 to 70 per cent hearing? From 50 to 60 per cent hearing? Less than 50 per cent hearing? Of those having less than 90 per cent hearing, what percentage shows purely a conductive defect? What percentage shows a perceptive defect? What percentage shows a mixed conductive and perceptive defect? What percentage shows evidences suggestive of otosclerosis?

From the standpoint of the practical reason for such an undertaking, namely the prevention of deafness, it is obvious that the school authorities would notify the parents of each child that showed defective hearing, and the parents presumably would consult their physician.

Incidentally, the results of such an investigation would be of value in determining the best seating arrangement of school children in the classroom, so that those with the greatest impairment of hearing would be seated nearest the teacher.

It is beyond the purpose of this paper to conjecture what benefit would result, for example, from the timely removal of tonsils and adenoids in the cases showing conductive hearing defect, or from the results of antiluetic treatment in the child with hereditary syphilis. It is purely speculative to attempt to estimate how much good could be accomplished by early medical or surgical intervention. The wisdom of undertaking such early treatment, however, is obvious.

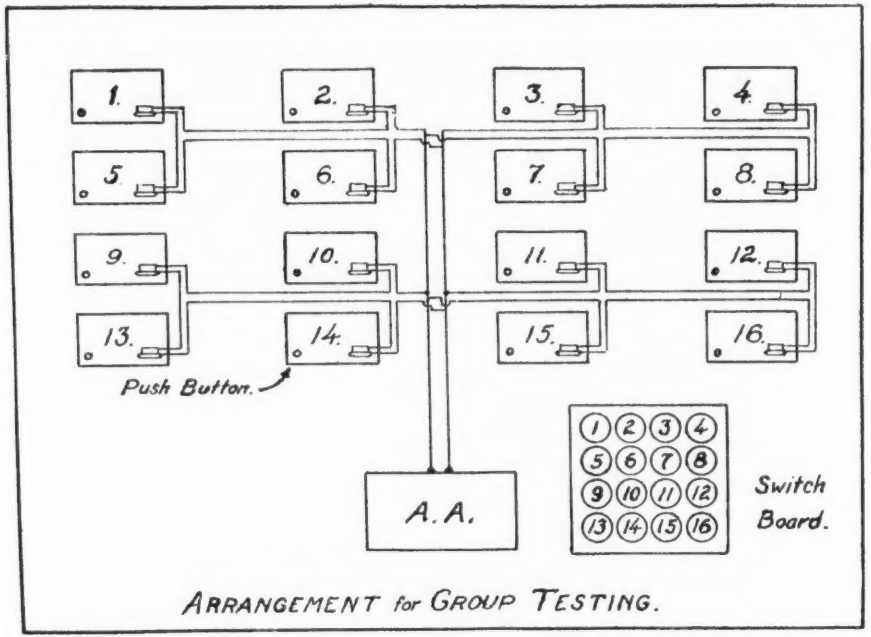


Fig. 1. Arrangement of apparatus for testing a group of sixteen.

HEARING CHART FOR GROUP TESTING.

Group No. _____ Date. _____

Grade. _____ School. _____

Operator. _____

				Right Ear.					Left Ear.				
				Upper	Percent Hearing.				Opper	Percent Hearing.			
#	Name.	Age.	Sex.	Limit	4096	1024	256	64	Limit	4096	1024	256	64
1													
2													
3													
4													
5													
6													
7													
8													
9													
10													
11													
12													
13				16500	90	85	78	68	17000	92	85	75	65
14													
15													
16				8200	55	84	92	95	7500	52	81	90	95

Fig. 2. Recording Sheet for group of sixteen.

AUDITORY CHART.

Name_____ Date_____

How old when hearing impairment first observed?_____

Describe onset_____

Hear better in presence of noise or in quiet room?_____

Any deafness in family?_____

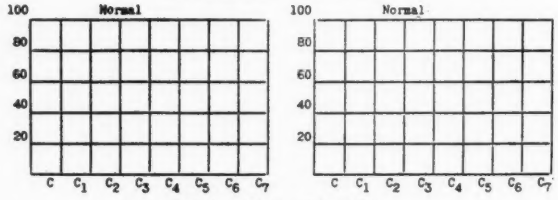
How hear over telephone?_____, radio?_____

Tinnitus?_____

Measurements with Audio-Amplifier.

RIGHT EAR.	LEFT EAR.
I. Amplifier.	
Voice, confused_____, distinct_____.	Voice, confused_____, distinct_____.
Heard best with uniform amp_____.	Heard best with uniform amp_____.
Low pass amp_____, High pass amp_____.	Low pass amp_____, High pass amp_____.
Whisper, percentage hearing_____.	Whisper, percentage hearing_____.

II. Percentage Hearing for Tones.



III. Upper Limits of Audibility.

_____d.v. _____d.v.

IV. Bone Conduction.

128_____, 256_____, 512_____, 128_____, 256_____, 512_____

V. Special Findings.

(Pitch and loudness of tinnitus, displacusis, tonal islands, etc.)

Diagnosis:_____

Comments:_____

Note: C – 64 d.v., C_1 – 128 d.v., C_2 – 256 d.v., C_3 – 512 d.v., C_4 – 1024 d.v
C_5 – 2048 d.v., C_6 – 4096 d.v , C_7 – 8192 d.v

Fig. 3. Chart for Individual Record.

LIII.

THE RELATION OF MINOR INJURIES TO THE NOSE DURING CHILDHOOD TO SUBSEQUENT DEFORMITIES OF THE ORGAN.*

By William Wesley Carter, A. M., M. D.,

New York.

An extensive and varied experience in correcting nasal deformities during the past eighteen years has brought under my observation many patients having marked deformities with more or less complete obstruction to nasal breathing, who would, in all probability, never have been so afflicted if the injury which disturbed the developmental balance and was responsible for the deformity had been recognized and properly treated at the time.

It is with a view to calling attention to and emphasizing the importance of regarding the slight injuries that the nose suffers so frequently during childhood as potential factors in the production of nasal deformities and obstruction to nasal breathing that I offer this communication. My observations are drawn purely from personal, clinical experience; my suggestions, therefore, will be for practical application, and I trust that they may be of some value in the field of preventive surgery of the nose, a field which, so far as I know, has been entirely neglected by rhinologists, and which is of great importance both from a cosmetic and functional point of view.

I will not enter into a minute discussion of the embryology of the parts involved, but as the origin and development of the nose is so intimately associated with the subject under discussion, reference to the embryologic and developmental facts that are distinctly of clinical value is essential in order that I may make clear my argument in favor of prompt and

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*Read at the annual meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society, Asheville, N. C., February 27, 1926.

proper treatment of all injuries to the nose of the child, with a view to limiting the number of operations which we are called upon to perform in later years for the relief of nasal obstructions and deformities due to neglect of the original injury which occurred in the early stages of the development period.

The nose at the time of birth is almost flat, even the tip is raised only slightly above the plane of the cheeks, and there is no elevation to the bridge. If this immature, undeveloped organ be compared with the fully developed, prominent nose of an adult, and if we compare their respective elevations above the facial planes, we can realize the great amount of work that must be accomplished by the developmental forces residing in the infant's nose. Let us assume that the bridge of the nose is raised by these forces acting through three piers, namely, the two nasal processes of the superior maxillæ and the septum. These forces must act in unison, with uniform vigor, and the resistance to be overcome must be the same on both sides; otherwise there will be distortion, resulting in deformity and obstruction to nasal breathing.

In the nose there is normally an unequal growth of related parts, and this accounts for the changes which occur in this organ between the time of birth and puberty, when the nose has usually assumed its adult proportions and has acquired its racial characteristics to a considerable extent. The large amount of clinical material that has passed through my hands has enabled me to form a fairly accurate estimate of the origin, the direction and the relative power of these several development forces. This knowledge has been greatly augmented by a study of the dried skulls in the Cornell University Museum. The conclusions reached have been drawn from observations of the results accomplished by these developmental forces and by the evidences of strain which certain parts of the nasal framework have suffered during the process of development. In infancy the septum is straight and usually remains so up to the age of seven years, while it is deflected in 75 per cent of white adults. In addition to deflection, there are often found spurs at the junction of the septal cartilage and the vertical plate of the ethmoid with the vomer. Now

these deviations from the normal which have made their appearance since infancy are not merely adventitious happenings; they are the evidences of mechanical strain, written in ossified characters so plainly that he who runs may read. All of my studies, both clinical and osteologic, show that the nasal bridge is raised chiefly by forces transmitted through the septum. The embryology and the developmental history of the vomer indicate very clearly that these forces, to a large extent at least, reside in this bone and from this source are so directed that they raise the flattened nasal bridge of the infant into the relatively more prominent position which it occupies in the adult.

The strategic position of the vomer is impressive, for it is wedged between the base of the skull and the arch of the hard palate, which furnishes an unyielding point of resistance for the growing septum to rest upon, and determines its direction of expansion upwards and forwards, where much of its force is expended in raising the nasal bridge. The septum, therefore, is under considerable vertical pressure, and at the same time it is confined by the more or less rigid walls of the nasal box. If it is thrown out of the vertical by any force it cannot recover itself, and the deviation, imperceptible at first, becomes a marked deflection as growth proceeds. At the same time, if the subject is a young person, the nasal bridge, deprived of the normal vertical action of this important development force, will retain its infantile characteristics and be broad and flat. The same effect is produced by any injury or disease, such as syphilis, which retards the growth or destroys the vomer.

In the embryo the vomer is represented by the vomerine cartilage, which is covered on either side by a membrane. This cartilage is an embryonic structure. With the exception of that portion which later becomes the septal cartilage, it is absorbed, the vomer being developed in the following interesting manner from the membrane which covers it on either side. At about the eighth week of fetal life osteogenetic centers make their appearance in the posterior part of the membrane which covers either side of the primitive vomerine cartilage. Ossification begins posteriorly and proceeds anteriorly. Two

plates of bone, therefore, formed as these membranes ossify begin to unite posteriorly at about the third month, and their union gradually extends forward, the vomerine cartilage between the two plates being absorbed, but to a certain extent pushed forward, as the union between the two plates of the vomer proceeds. This pushing forward of that portion of the vomerine cartilage, which later becomes the cartilaginous septum, assists, but only in minor degree as compared with the more direct action of the vomer, in raising the nasal bridge. The two plates of the vomer do not become united until puberty, and it is very suggestive that this period coincides with the full development of the nose. The anterior border of the vomer extends upward and backward and corresponds to the prospective contour of the adult nose; the forces, therefore, that spring from the vomer act at right angles to this contour, the most effective direction that they could take. Normally the two plates of the vomer, at the anterior edge of this bone, do not coalesce, but form a groove, into which the septal cartilage and vertical plate of the ethmoid fit. Now it is along this suture that we find positive evidence that the septum was under great vertical pressure during the development period. Spurs on both sides caused by the spreading of the upper edges of the vomer, overriding of the septal cartilage and vertical plate of the ethmoid, or angular deflection of the septum at this point were observed in 90 per cent of the dry skulls I examined, and I feel quite sure that we meet these deviations from the normal as frequently in our clinical work. A winged spur of greater or less proportions on both sides of the septum, due to compression of the tonguelike sphenoid process of the septal cartilage, where it is wedged in between the vomer and the vertical plate of the ethmoid, is nearly always present in adults and is positive evidence of the great vertical tension under which the septum labors during the development period. It can be readily understood, therefore, if the septum is thrown out of the vertical at this time of life, if it is fractured, if it is destroyed by disease or removed by operation (submucous), a deformed nose is certain to be the result.

If there is complete failure of union between the plates of the vomer, not only is the nose broad and the entire bridge depressed, but there is a sulcus between the two nasal cavities

which practically divides the nose into two lateral halves and constitutes the deformity known as wolf nose. This condition is not very common; I have seen only five cases.

When one considers the delicate balance of the forces employed in the development of the adult nose, it is unnecessary to emphasize the fact that the trivial as well as the more severe injuries which the nose receives during the development period should receive expert attention as soon after the accident as possible. X-ray plates of nasal fractures are seldom satisfactory; this is true especially if the patient is a child.

Inspection is of value if the case is seen before much swelling has occurred. The best method, however, for determining the nature and extent of an injury to the nose is by bimanual palpation, which is practiced in the following manner: A suitably shaped instrument, such as the blunt, flexible end of my submucous elevator, is introduced into the nose and held with one hand closely against the roof of the nasal cavity, while the forefinger and thumb of the right hand palpate the entire nose in an effort to discover crepitus or abnormal mobility. This is sometimes very difficult to determine, for blood crepitus and emphysema may very closely resemble bone crepitus. If the patient is not seen immediately after the accident the tumefaction of the tissues may mask the extent of the injury. When in doubt it is better to employ antiphlogistic measures for a day or two until the swelling has subsided, and then make a thorough examination, if necessary under a general anesthetic. In the case of children, and this is the class of cases we are now considering, it is usually necessary to give a general anesthetic in order to make a comprehensive study of the situation and to apply the treatment I am about to advocate.

An injured nose was formerly cleansed and mobilized by means of a piece of adhesive plaster drawn tightly across the bridge. This was worse than no treatment, for depression was favored by causing the fractured parts to override. Today we are seeing the results of this mismanagement. If the patient was young when the injury occurred and he has reached adult life, we have to do with a developmental deformity; while in the case of a neglected or improperly treated injury to the

nose of an adult we have traumatic deformity, due to the mechanical displacement of the segments of the nasal arch, the interference with the developmental forces here being a factor of minor importance, as these had already expended their energy at the time that the injury was received. In both of these instances a surgical operation is necessary for the correction of cosmetic and functional defects, both of which might have been avoided if proper treatment had been administered at the time of the injury.

When it has been determined that there is a fracture by the methods of examination that I have outlined, the fractured parts should be brought into apposition and the nose lined up in its normal shape and position by means of my fracture forceps, one blade of which is placed on the outside of the nose and the other within the nasal cavity. In estimating whether or not the fracture has been properly set, we must depend upon the sense of touch and upon the appearance of the nose. The X-ray in children does not give accurate information, but it is sometimes of great assistance in adult cases. After setting the fracture, if the child is over twelve years of age and is amenable, I use the bridge splint, with which you are all familiar, but in young children this instrument is not applicable, so here I make use of my gold wire splints in the following manner: I take two wood tongue depressors, whittle them to fit the nasal cavities, and then wind around them, in a spiral, a piece of No. 10 gold wire about six inches long. When the wooden obturators are withdrawn we have two spiral wire splints that accurately fit the nasal cavities. These splints are then covered with vaseline, and by means of a pair of narrow, long bladed forceps, are introduced into the nasal cavities, care being taken to have the anterior edges of the splints fit into the nasal vestibules, so as to prevent them from being dislodged. The nasal cavities should be cleansed once or twice a day with an alkalin solution. The splints should remain in the nose for ten days.

We all know that patients upon whom intranasal operations have been performed complain more bitterly of the packing than they do of the operation. The use of these splints adds greatly to the comfort of the patient, as he is able to breathe

through his nose during the entire period of convalescence. Furthermore, the nasal cavities can be kept clean and treatment applied much more readily.

In conclusion, I will say that I am convinced, from a study of the dynamics of nasal development, that many of the deflected septa and deformed noses seen in adult life could have been prevented if the injuries to the nose, received during the development period of life, had been properly treated at the time.

24 East Forty-eighth St.

Fig. 1.

Fig. 2.

CASE 1.

Developmental deformity, due to traumatic injury in early life.
(Corrected by transplantation of conjoined bone and cartilage.)

Fig. 1.

Fig. 2.

CASE 2.

Developmental deformity due to fall from crib at two years of age.
(Corrected by transplantation of bone and cartilage.)

Fig. 1.

Fig. 2.

CASE 3.

Infantile type of deformity due to traumatic destruction of bony septum in early life.

Congenital deformity—Wolf nose.

LIV.

MUCOCELE OF THE FRONTAL SINUS.

By Louis Daily, M. D.,

Houston, Texas.

Rose Walls, female, colored, age 51.

Family history negative.

Previous history: Measles, mumps, chills and fever.

Present complaint: Protrusion over her left eyebrow. One year and a half ago she fell against a door and received a blow over the forehead and she dates her trouble from that. For six months she suffered severe pain, robbing her of sleep; the swelling over her forehead was increasing. For the last year she has had no pain, but the swelling is increasing.

Examination, July 1, 1925: Firm round protuberance over left frontal sinus; on hard pressure fluctuation could be elicited. The bone over the center of the swelling was gone, except for some projections extending from the upper border. Left eyeball displaced 1 cm. downward and one and one-half cm. out. Movement of left eye limited up and in.

Rhinoscopic examination negative.

General physical examination negative; Wassermann negative; X-ray showed an enlarged left frontal sinus encroaching on the sphenoid and ethmoid sinuses.

Operation, July 8, 1925: Local anesthesia; 2 per cent novocain injected around the anterior and posterior ethmoid nerves at the inner angle of the orbit. The infraorbital nerve blocked at the infraorbital foramen. The maxillary branch of the trigeminal and the sphenopalatine ganglion reached through an injection under the angle of the zygoma.

Operation: Modified Killian incision below the eyebrow which went through the mucous membrane of the sinus, which was filled with thick, gelatinous, tenacious, slate-colored mucus; this was evacuated with the suction tip. Most of the anterior sinus wall and the floor of the sinus were absorbed. The

posterior wall was intact. The sinus was enlarged at the expense of the anterior and inferior wall. The remains of the anterior wall were left undisturbed; the nasofrontal duct was entirely closed. In cleaning out the cavity near the sphenoid, an opening was made into what seemed probably the cranial cavity, and for this reason it was thought best to drain externally and not through the nose for fear of infection. The cavity was packed with iodoform gauze and drained along the inner orbital angle; the rest of the wound was sutured. The wound drained profusely and she complained of pain. On July 30th, under local anesthesia, an intranasal opening was made into the frontal sinus, with the Halle technic. The pain immediately disappeared, and the external opening promptly closed, and the drainage through the nose was profuse for a few weeks. It gradually diminished and ceased entirely in eight weeks. Bacteriologic examinations of the contents of the mucocele showed no organisms present.

At present, as you will notice in the picture, her eyeball has returned to its normal position, and the movements of the ocular muscles are good. She still has monocular vision with the right eye fixing, but a vertical diplopia, with the right eye higher, can be elicited. Her vision before the operation was 20/40 and is now 20/20.

Remarks: The striking things in the history are the absence of diplopia and the history of diplopia. This is probably due to the patient's low intelligence. At the time of the examination she had monocular vision with the image of the left eye suppressed, but diplopia could be elicited.

The second point in the history is the unusual rapidity of development, which probably accounts for the severe pain.

In the convalescence of the case is to be noted the shrinking of the enlarged cavity. The bony protuberances were left undisturbed and the bone receded to its normal position; after the cavity is drained it is not necessary to remove the overlying bone. In the surgery I would like to point out the advantage of combining the external frontal with a Halle intranasal operation; this can be done at one sitting or a week or two apart. The advantage is the conservation of the middle turbinate,

the permanency of the opening left after the removal of the agger nasi.

Dabney of Washington, in a review of the literature on mucocele in 1921, collected 75 cases, including two of his own. Since then the number of reported cases has been brought to about 100. Various theories have been advanced for etiology, from a chronic sinusitis to a cyst in the sinus in the mucosa. Traumatism is a very prominent factor in precipitating the case.

647 Kress Bldg.

Fig. 1.
Photograph taken before operation.

Fig. 2.
Photograph taken after operation.

Fig. 3.

Fig. 4.
Skiagraph taken seven months post operative, showing sound in nose, opening still patent.

LV.

RADICAL MASTOID SURGERY. END RESULTS.

By Ernest M. Seydell, M. D.,

Wichita.

The constant changes in the attitude of the otologists relative to the value of the radical mastoid operation in the treatment of chronic suppurative otitis media seem sufficient evidence to the writer that the results obtained by this procedure have not been universally satisfactory.

In writing this paper it is not my purpose to review extensively the literature on this operation, but rather to touch only on the important phases of the subject, with the object of bringing out a frank discussion covering this troublesome condition. The end results obtained in radical mastoid surgery are one of the causes of its present unpopularity. Some of this unpopularity is deserved and cannot be avoided. However, much of the discredit which has been cast upon it is not merited and can be obviated. The objection most often met with from the patient's standpoint, and the one which also meets with the disapproval of many medical men, is that the discharge continues in many cases. Another point which is often brought out against the operation is that the patient must be under the care of an experienced otologist: First, through a long period of after treatment, and that subsequently he is obliged to visit his physician at stated intervals for observation for at least a year. Again, many of the cases so treated have considerable loss of hearing.

In considering the first point, the continuance of a discharge from an operated ear, it becomes necessary to divide our cases of chronic suppurative otitis media into several classes. The following classification is satisfactory for this study:

1. Nondangerous.
2. Dangerous.
3. Cholesteatoma.

The nondangerous type usually presents a central perforation of the tympanic membrane, with a chronic suppuration of the tympanic mucosa and eustachian tube. In these cases the mucous membrane of the middle ear cannot be completely removed, and since it is not always possible to close the eustachian tube a continuance of the discharge is frequently found. The radical operation is not indicated in this type of chronic suppurative otitis media, and much discredit has been cast upon the operation by surgical intervention in these cases.

In the dangerous ears, where bone necrosis exists, the results depend on the thorough removal of the necrotic bone and careful after treatment, in my mind, the latter being almost as important as the former. I firmly believe that all advantages of a perfect operation may be lost by carelessness or lack of knowledge in the treatment of the operated cavity.

Richards points out six reasons for operative failures: First, failure to lower the facial ridge to its absolute limit; second, failure to remove the serrated fringe of bone representing the anterior margin of the facial ridge back to the descending limit of the facial nerve. This removal should be carried down to the level of the hypotympanic floor. Third, failure to lower the floor of the bony auditory canal down to the level of the hypotympanum. He states that unless these three steps are taken it is impossible to get at a mass of cancellated structure posterior to the promontory, which is always diseased in suppurative otitis of the chronic type. This cancellated structure is peculiarly hard and firm and shows little tendency to throw itself off by process of caries. Fourth, failure to shave down the convexity of the anterior bony canal wall so as to increase the anteroposterior axis of the cavity and, in conjunction with No. 2, to render the tympanum more accessible. Fifth, failure to remove the annulus tympanicus completely and the lip of bone overhanging the mouth of the eustachian tube. Sixth, failure to remove the attic wall.

I must confess that I do not follow this radical a procedure in all of my cases, but am rather inclined to clean out the mastoid cells, antrum and attic wall, and lower the facial ridge, after which I thoroughly inspect the middle ear, and if conditions seem to require it, proceed as radically as I deem neces-

sary in the case in question. On the whole, I rarely curette the middle ear.

In the cholesteatoma, as in the chronic suppurative cases due to bone necrosis, the results depend upon the removal of the causative lesion, the cholesteatomatous mass. In addition, the underlying bone must be carefully searched for extensions of the cholesteatoma into the bone, and these extensions must be thoroughly curetted.

I realize, however, that a few aural surgeons recommend leaving the matrix, on the supposition that it may become converted into a healthy epithelial lining. I have had recurrences even after a very thorough removal of the cholesteatoma.

The object of the radical mastoid operation in any given case is to remove the causative factor, and after this has been done, to promote the complete epidermization of the mastoid cavity and middle ear. The epidermization should be accomplished as speedily and with as little distress as possible. In a general way, this is brought about either through using packs in various ways or by the use of Thiersch grafts, applied either primarily or secondarily.

There are almost as many methods used in the after treatment of radical mastoid operations as there are aural surgeons. Where no grafts are used, any method that will insure a smooth growth of granulations over which the epithelium may creep, using care not to allow an overgrowth in any portion of the cavity, will insure success. An overgrowth of granulations might cause septi to form, which in turn could wall off a portion of the cavity, the same remaining infected.

Those who favor Thiersch grafts state that healing is very much more rapid and the after care less painful. Here again each operator has his own method of applying the grafts. So far as I can learn, the primary graft is more popular in the United States. The grafts may be cut into small pieces and then carefully placed over the surface of the mastoid cavity, and when this plan is used, I think, the Pontopiddaus method is the easiest to carry out successfully. Pontopiddaus spreads the graft, cut surface down, on a spatula. The graft is then transferred to a layer of gauze, which has been coated with a

thin layer of adeps lanæ. The graft and gauze are then cut into suitable pieces and applied as desired.

The method devised by Mosher should appeal to those who desire a more rapid technic. Mosher uses a four ply gauze tape 1½ inches wide which has been dipped in paraffin. Before it is put to use it is softened in warm water. A cone is formed of the paraffin tape. This cone is inserted into the mastoid cavity and moulds itself accurately to its conformation. The mould is then removed and covered with the graft. It must be cut off flush with the surface of the mastoid and not project. If a meatal flap is used it is tucked in place back of the mould and its cavity filled with ordinary gauze. The mould is left in place for from five to ten days. In removing it the gauze is removed and the sides of the basket are collapsed.

The majority of the statistics published relative to the percentage of dry ears in radical mastoid operations for chronic suppurative otitis media are valueless, as they have been compiled from all types of cases. To be of value it would be necessary to study each case according to the type to which it belongs.

I believe that in most cases of bone necrosis or cholesteatoma, which receive proper care, one may expect to get dry ears, except where a tubal suppuration exists, and it is impossible to close the tube, or where the middle ear retains its lining of cylindrical epithelium, in other words, where complete epidermization does not take place.

As to the second objection—it cannot be denied that considerable time is required for postoperative treatment and that prolonged observation is essential. We have all had cases in which a total epidermization has taken place in three weeks, either with or without Thiersch grafts. Many cases require much longer and some must be reoperated before a cure is established. All patients upon whom a radical mastoid operation has been performed should visit their physician at least every sixty days, during the first year subsequent to the mastoid operation. Even though the ear is dry, products of desquamation accumulate in the mastoid cavity and produce pressure on the underlying epithelium, causing a destruction of the same. Infection may then take place, with the formation of

an ulceration and granulation tissue. If seen early, the localized areas of ulceration are easily controlled. Even though we admit that these things are true, it does not follow that we have not benefited the patient by performing the radical mastoid operation. The real reason for the operation is neither to stop the suppuration nor to increase the patient's hearing but to prevent intracranial complications. If, in addition to this, we have relieved the patient of a troublesome headache or vertigo we can safely say the operation was justifiable.

The third objection was that of loss of hearing. In performing a radical mastoid operation the patient's hearing may be injured in a number of ways. Among these may be mentioned trauma to the internal ear, destruction of the ossicular chain and the formation of heavy scar tissue in the middle ear. I have always felt that a severe curettage of the middle ear favored the formation of the latter.

In a general way, the degree of deafness following the radical mastoid operation depends upon a number of factors: First, the amount of hearing present at the time of the operation. If the hearing is only slightly affected there will be a loss of hearing. If considerable deafness exists, the hearing may improve. Second, the type and extent of the lesion for which the operation has been performed, as where disease has produced a destruction of the ossicular chain. Third, the operation itself and its after care.

POSTOPERATIVE RESULTS IN THE EARS OF CHILDREN.

A general review of the literature reveals the fact that in children there is a greater loss of hearing than in adults. The younger the child, the greater the loss of hearing. Therefore every effort should be made to relieve the condition by local therapy before resorting to the radical operation. Finally, the radical mastoid operation should never be considered from the angle of expecting an improvement of hearing.

Before closing, I wish to briefly touch on the results of the radical operation on the life of the patient. First, we must consider the danger of the operation and especially the fact that latent complications may manifest themselves following the operative trauma.

A careful examination of the patient prior to the operation will do much to eliminate these postoperative complications.

Can we promise the patient that he will have no intracranial complications when none exist prior to the operation? I think this question can be answered in the affirmative after making the following reservation: The patient will be safe, providing he does not neglect his ear condition. Lange states that he has not found in the literature any dangerous complications or deaths following the radical mastoid operation.

Fabry, however, reports a death from brain abscess, following a recurrence of a cholesteatoma. The patient was a four-year-old child, who was taken away from the hospital fourteen days following the operation and received no further treatment until twenty years later, when he presented himself with a brain abscess well advanced.

Henius and Schousboo critically examined the records, and subsequently the patients on whom a radical mastoid operation had been performed at the St. Joseph Hospital at Odense covering a period of twenty-two years. During this time 301 patients were operated upon. Nineteen of these had a bilateral operation. They were able to trace 234 patients, none of whom died from mastoid disease or any of its complications. Their inability to trace 54 patients makes it impossible to draw exact conclusions from these statistics, yet the fact remains that in the 234 patients traced none died of mastoid disease during the period of 22 years.

CONCLUSION.

1. The radical mastoid operation should not be employed in the nondangerous type of chronic suppurative otitis media.

2. In the dangerous type every means should be employed to remove the pathologic conditions before resorting to the radical mastoid operation. Where the disease is limited to the attic a meatal operation, as advocated by Dr. Tobey, should be considered.

3. If a cholesteatoma is the cause of the chronic suppuration, nothing short of an operation will bring about a permanent cure. If this condition is localized in the attic, a meatal operation should also be considered.

4. The radical mastoid operation should never be performed with the object of increasing a patient's hearing.

5. A dry ear may usually be expected, but it is only of secondary importance.

6. The patient may be assured that if no intracranial complication exists at the time of the operation, none will later arise, providing proper after treatment is instituted.

7. Patients must be seen by their otologist from time to time for several years subsequent to the operation.

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LVI.

THE CURE OF CHRONIC SUPPURATIVE NASAL CONDITIONS BY DRAINING THROUGH THE MAXILLARY ANTRUM.

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It is not my purpose to write a thesis on causation and symptomatology of all the diseases of the accessory sinuses of the nose; but rather to summarize some of the observations made during a quarter century of active practice in treating diseases of the nose and throat. For the first fifteen years of this period I followed the lead of such men as Chiari, Hajek, Gruenwald, Lambert Lack, St. Clair Thompson, Mosher and others, in diagnosing diseases of the various sinuses from the clinical symptoms and physical signs, elicited by transillumination, irrigation, etc. By these painstaking methods considerable progress was made in diagnosis and treatment.

During the last ten years we have added to the foregoing methods of diagnosis the use of the skiagram as a routine in all chronic nasal conditions. It is by means of this modality our former belief, that the majority of cases of chronic nasal catarrh resulted from suppurative ethmoiditis, gave way to the knowledge that it is the maxillary sinuses that are the offenders in the major number of these cases; and this is a logical conclusion, if we take into consideration its location and anatomic relationship to the other sinuses. There is no doubt that in early childhood, and even in adult life, in practically all cases of common, socalled colds, when long continued, or oft repeated, the maxillary sinuses participate in the pathology, and because of their dependent position are the last to clear up after acute attacks. If deflections or deformities of the septum add to the natural defective drainage from the antrums, they are prone to remain infected with the resulting degeneration of their lining mucosae into mucous polypi, cysts and suppurations which, I believe, cause

the majority of the chronic nasal catarrhs. It is easy to comprehend that although the ethmoiditis, which was the original source of infection to the antrum, is temporarily cured with or without local treatment, reinfection may occur, and polypoid degeneration from overflow or pus or acrid secretions from the chronic antrums results. These are the cases which seem to have short periods of freedom from nasal discharge with no apparent physical signs of disease in the nose, but which light up fresh symptoms upon the slightest exposure. In my early experience it was the custom to attack the ethmoids whenever there was a suspicion of disease in them manifest by polypus formation or enlargement of the anterior end of the middle turbinate. In the more exaggerated of these cases, mucous polypi so fill the nose that they project, not only from the anterior nares, but hang down behind the soft palate into the pharynx. The common practice twenty-five years ago in these cases was to snare off the polypi, only to have them recur in from six weeks to a few months. As we advanced a step in the treatment, a complete ethmoidectomy, including the middle turbinate, as advocated by Sluder, has been performed, or an exenteration of the ethmoids leaving the middle turbinal as advocated by Mosher and others. This treatment gave permanent cure in many cases, and more prolonged relief in others not cured, than did the method of merely snaring off the polypi.

It was found that in a great many cases, even after a thorough exenteration of the ethmoids, the polypi recurred in more malignant form than before. It is exactly in this type of cases, in our more recent experience, that we find the chronic maxillary sinusitis plays a very important part. It matters not whether the disease originates in the maxillary antrum, or whether it becomes secondarily infected from a previous ethmoiditis, the effect upon the progress of the disease is the same in these cases of recurrent nasal polypi.

To establish drainage from the antrum by making a free opening into the nose at the most dependent part seems a rational treatment, and in our hands has proved most satisfactory, not only in curing the discharges from the antrum, but for drainage of the ethmoid cells. We have had numer-

ous cases of mucous polypi originating in the ethmoids clear away after drainage of the antrums. Of recent years our nasal surgery has swerved from frequent attacks upon the ethmoid labyrinth to the surgery of the antrum and the septum. Careful observation has led me to believe that there are very few cases of chronic suppurating sinuses that are not attended by some deformity of the septum, either deflection or exostoses from it, causing mechanical irritation and obstruction to ventilation of the sinuses. Therefore, correction of defects in the septum has become a first consideration in nasal pathology, and it is surprising how many cures are effected by this treatment alone. This is especially true in cases of diseased sphenoid and posterior ethmoid sinuses. All rhinologists are agreed that preservation of the healthy mucosa is essential to reestablish the physiologic function of the nose.

What then should be the treatment for cases of chronic discharge from the nose, either with or without polypus formation? We first of all correct any deformity of the septum which interferes with proper ventilation or drainage to any of the accessory sinuses. If the skiagram shows cloudy antrum this is drained by a radical Caldwell-Luc operation, which I believe to be the most rational and uniformly successful operation on the antrum. I shall not go into detail of the operation any further than to say that we operate under local anesthesia. We make the opening through the canine fossa, just large enough to get a good view of the whole mucous lining, and of sufficient size to allow thorough removal of mucous polypi and pus pockets in the mucous lining of the antrum. Through this opening we remove the whole inner antral wall from the floor up to the attachment of the inferior turbinal and the inner antral wall. I have observed that this angle is a frequent seat of mucous polypi. The inferior turbinal is not disturbed unless it be in cases where there is polypoid degeneration, or great hyperplasia of the mucosa, in which event a part of the lower border is excised so as to maintain drainage.

The wisdom in choosing the canine fossa for the operation on the antrum will be clearly justified by the lantern slides which follow. I have given up entirely the method

of going through the nasal route for draining chronic antrums, for the reason that the operator cannot see what he is doing and that the internal operation in my hands is not as effective, and is attended with more pain than the external method.

The after treatment is very simple. We pack the cavity with a single strip of iodoform gauze for twenty-four to thirty-six hours, leaving one end in the gingival wound. When the packing is removed the wound is allowed to close without sutures. We seldom resort to any irrigation of the sinuses in the after treatment. We order saline irrigations for the nose, once or twice a day, and we swab the nose and sinus through the nasal opening once a day for ten days or two weeks. After that time we prescribe some bland petrolatum mixture containing a little menthol and oil of eucalyptus for home use. It is surprising how quickly those cases heal by this method of treatment.

I do not wish to convey the idea that all chronic suppurative conditions of all the nasal accessory sinuses can be cured by drainage of the antrums in the manner outlined above, but I do want to emphasize the fact that this method is applicable to and will cure a great many cases of chronic suppurative ethmoiditis with polypus formation that cannot be cured without draining the maxillary antrums.

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LVII.

CONGENITAL OSSEOUS OCCLUSION OF THE POSTERIOR CHOANÆ—REPORT OF CASE.

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and
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The fact that very few records of congenital osseous occlusion of the nares appear in medical literature does not necessarily suggest this condition is a rare deformity. Many authorities, including Richardson, Clark, White, Clay, Fraser and others, believe that this condition exists more frequently than the approximately 150 cases reported would indicate. They believe that the many infants who die in the first few hours of life would reveal many more cases of congenital osseous occlusion of the nares, which have passed into the records as asphyxia neonatorum. The wonder of most of these men is not that such a congenital anomaly exists as that the individuals should survive to that age in which diagnosis becomes easy. That the number of observed cases has increased in the last twenty years is proof that the pharyngologist is more careful in his examination and also attests to the more intelligent management of the infant by the obstetrician and hospital corps.

Otto reported the first case of congenital osseous occlusion of the choanæ in 1830. This was discovered in the examination of a fetus. It remained to Emmert to make the first clinical report on the living subject in 1851. In 1886 Hubbel gave a résumé of all cases reported at that time, numbering seventeen, including his own. Upon these many operations had been attempted with varied success.

Hubbell used a hand drill and procured a good opening in bilateral occlusion, but these soon closed with granulation tissue and required a secondary operation. Blocked tin tubes were inserted and worn for six weeks with ultimately good results.

In 1898 Clark reviews 20 cases in literature, including his own, which he trephined with good results. Schmiegelow, in 1904, gathered 61 observed cases, most of them osseous. Fraser, in 1910, estimated the reported cases at 108, while Pfingst, in 1914, computed the cases in literature as considerably over a hundred. White, in 1919, estimated the probable frequency of this deformity as less than 150. Since then we have not been able to collect from the more recent literature more than a dozen to add to the above number.

The fact that osseous, cartilaginous, osseomembranous occlusions are congenital and that it is not any longer necessary to defend such a contention was well established by Richardson in 1914, in his collected reports of Otto, Luschka, Bitott, Gross and many others of later date. He further states that he believes the obstruction takes place in utero and is not due to inflammatory reaction or pressure. However, on the other hand, heredity does not seem to be an important factor, and Richardson calls Lang to task for trying to establish this contention, as he did not thoroughly enough investigate all the cases included in his report to establish the claim of heredity. Jacques believes that syphilis plays an important rôle as a predisposing factor in this condition, while Mauret and Cozejust would also add tuberculosis.

Congenital occlusion of the nares may be osseous, cartilaginous or membranous. Osseous occlusion seems to be of the greatest frequency, judging from Fraser's report, in which he collected 89 cases of bony obstruction and but ten of bone and membrane. Bilateral occlusions, according to the more recently collected records, are more common in the ratio of 3 to 1. Clark, in 1898, found more unilateral cases reported, but Richardson, in 1914, found that the bilateral predominated in the proportion mentioned above. Such an increase in the reported cases of bilateral occlusion in the last 20 years is occasioned, we believe, by the more intelligent management of the newly born infant by the physician and the more improved hospital methods. When unilateral occlusion does occur, it is found on the right side more frequently. Richardson found females more frequently affected than males.

While most of the occlusions are osseous, the obstruction may be osseomembranous, membranous or cartilaginous. The

only case of complete cartilaginous occlusion was reported by Onodi. Most of the deformities are found about a millimeter anterior to the posterior border of the hard palate. The anomaly may be found elsewhere, according to the classification of Kayser—(1) Intranasal, (2) marginal, (3) extranasal or retronasal—that is, practically within the pharynx. Of these, marginal atresiæ are the most common. Anterior atresiæ of osseous nature are extremely rare. The deformity may be complete or incomplete.

Histology of the bony occlusion has been well summarized by Hockheim: "In structure it corresponds to the hard palate. It consists as hard palate of a centrally placed plate of bone, which on the one surface was lined with mucoperiosteum corresponding to that of the nasal cavity and on the other with the mucoperiosteum corresponding to the postnasal cavity, modified in the respect that in this are found glands, and the epithelia are columnar instead of flat epithelia."

Several theories have been advanced regarding the etiology, but most of them may be summarized as failure to live up to the embryologic requirements. Richard collected the theories as follows:

Luschka—The obstruction to be an extension upward and backward of the horizontal plate of the palate bone.

Kundrat—An extension inward of the vertical plates of the palate bone.

Hopman—An extreme degree of the occasionally observed nasal asymmetry.

Bitot—A separate and independent bone, as indicated by the sutures he observed.

In most of the obstructions observed the specimen has been more or less mutilated in removal, and until a sufficient number of unmutilated specimens have been observed we are at liberty to take our choice of the above theories.

The symptoms of congenital occlusion of the nares must be divided into those of the newborn and those of the adult. As has been implied, it is the consensus of opinion among modern rhinologists that the condition is more frequently overlooked than diagnosed. In the newborn the symptoms are very much

those of asphyxia neonatorum, the struggle to establish nasal respiration. The struggle is relieved by crying. As soon as the infant has obtained sufficient air the crying subsides and the struggle to get air through the nose is resumed. The infant is unable to nurse and must be fed by hand. On account of the inability to take food naturally and the repeated cycles of crying and asphyxia, malnutrition soon supervenes. Diagnosis may be facilitated by filling the nose with water and noting that the fluid does not reach the pharynx and has no effect on respiration. Also the nose can be filled to overflowing.

The most striking symptom in the adult is that of the absence of nasal respiration. There is atrophy of the nasal structures and absence of smell and taste. The choana is filled with glairy white mucus. Eczema of the alae is common. Tears flow out of the nose when crying and the patient gives a history of not being able to sneeze. The timbre of the voice is that of complete nasal obstruction. There is a catarrhal otitis media, although in many cases no change has been noted in the ears. The facial expression is not so marked as in complete nasal obstruction due to vegetation in the nasopharynx and pharynx. The nasolabial folds are not so pronouncedly drawn nor the mouth so widely agap. Asymmetry of the face is not a constant symptom, even in unilateral obstruction. Haag contends that unilateral nasal obstruction does not cause asymmetry of the face, as he found asymmetry in cases of bilateral obstruction. Richardson found only thirteen cases of asymmetry in his report. Haag contends that the asymmetry is not due to the obstruction, but to the same fetal condition that caused the deformity. Highly arched palate is not always a constant finding. Haag collected 68 cases from the literature and found 15 cases of highly arched palates, 6 normal and no mention made in the others. His own three cases showed highly arched palates. Kahler's nine cases all presented highly arched palates. Lang saw six cases, all with high palates. Fraser, in 1910, in the presentation of his case with measurements summarized the arguments for and against the theory that mouth breathing caused highly arched palates. Siebenmann, Frankel, Grossheintz, Haag and Buser believe that a highly arched palate is only part of a congenital high and narrow formation of the facial skeleton—that is, of leptoprosopia. Black, Körner,

Waldow, Alkan and Bentzen are of the opinion that mouth breathing produces an abnormally high palate. The summary of this theory is given by Fraser:

". . . . on account of the pressure of the stretched cheeks on the lateral part of the alveolar arch and also because of the want of counterpressure of the tongue on the roof of the mouth. These two factors are, of course, due to the open mouth and in addition there may be a certain amount of pressure due to the impact of the air current on the roof of the mouth. There is one factor that may aid in the production of the high palate, and that is a degree of atrophy of the nose from disuse." Körner and Waldow call attention to the high palates reported in congenital bilateral atresia to support their claim, but as there is no mention of the palate made in more than half of the cases we must assume that the palates were normal. In Fraser's case of unilateral occlusion both maxillary sinuses were equal in size, which is against the theory of Frers, that the development of the nasal accessory sinuses is due to the pressure of the air currents in the nose. Kayser states that a true atrophic rhinitis has not been observed in these cases. Asthma is an unnoted symptom. The anemic appearance of the patient is no doubt due to the inability to breathe through the nose, as the volume of air respired through the nose is one-seventh more than respired through the mouth. Fraser noted a small canal about the middle of the bony plate nearer the septum which admitted a probe—the canal ended in a blind pocket. A similar condition prevailed in our case.

Treatment of this condition must be considered in regard to the age of the patient. In the newborn the treatment has been with most of the observers that of support until the patient had attained an age sufficient to warrant radical procedures or until the nasopharynx had developed sufficiently to allow the entrance of the index finger—this last precaution to prevent injury of the postnasal structures by the trephine. This is usually possible at about the ninth month. However, Richardson and Loeb believe that the operation may be attempted at a much earlier date, even as early as the first few days of the patient's life, thus avoiding the many weeks of struggling for existence and the period of malnutrition. This

last procedure seems reasonable. However, the difficulties of the operation would be greatly increased on account of the small space in which to work, but much less resistance would be met with in the obstruction than later. No report has come to our notice in which the attack on the obstruction was attempted at such an early date.

Operation on these obstructions in the adult has been either by trephine and cutting punch or the submucous route. Tubes, bougies and packs are necessary over an extended time in either method, but the time of the after treatment seems shortened through the submucous method, although this does not seem to compensate most authorities for the difficulties of the latter operation. Kirby recommends the removal of the posterior end of the septum with the obstruction by the submucous route. Most of the cases in which the obstruction was removed in the last few years has been submucously.

F. F., aged 12, female. Slightly anemic, but with no other general appearance of interest. History of never having been able to breathe through the right side of nose. Had adenoids removed in early childhood, but with no relief. Has never been able to clear the right side of the nose of mucus, which at times caused a slight eczematous condition about the ala. There was no asymmetry of the face and the palate was but slightly high. Examination of the nose showed the right side filled with glairy white mucus, which was removed by lavage. The turbinals were slightly atrophic and the mucous membrane paler than on the left side of the nose. A complete bony obstruction filled the posterior part of the choana. Unable to use a postnasal mirror, but believe the deformity to be at the posterior border of the hard palate. The plate extended from the floor of the nose and the septum outward to the lateral wall and backwards to the sphenoid.

About midway up and close to the septum was a small canal that would admit a probe. This canal extended toward the septum but ended in a blind pocket. Firm resistance was met in every part of the obstruction. The parents were not ready to submit the child to operation, so no further investigations were made.

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LVIII.

THE ROLE OF THE OPHTHALMOLOGIST AND OTO-RHINOLARYNGOLOGIST IN THE DIAGNOSIS OF DUCTLESS GLAND DISEASE.*

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INTRODUCTORY.

It is a privilege and a pleasure to address some remarks to this organization on the relationships that obtain between the hormonopoietic system and the more specialized organs of sense. Some interesting contributions have been made during the past fifty years by eminent representatives of your specialties toward a wider knowledge of the ductless glandular system, especially as it applies to diseases of the eye, ear, nose and throat. I need but call attention to the notable observations of such men abroad as Hirsch of Vienna, Uhthoff of Breslau, the distinguished ophthalmologist, Fuchs, and the celebrated von Graefe; and in our country De Schweinitz, Zentmayer, Ziegler, Friedenberg and, here at home, Dr. Grant Selfridge.

At the outset, in approaching any discussion of the field of endocrinology as it applies to special regions of the body, it is of paramount importance to preserve a certain poise or equanimity. I like to use, in this connection, the admirable phrase that I first heard employed by that great authority on nutrition, Dr. Mendel, namely, "the prepared open mind." Much sagacity and wisdom lies behind this well chosen phrase. In the field of endocrinology, as in all newer realms of investi-

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gation, it is of the utmost importance to possess, as far as possible, a "prepared mind," one of such compass and comprehensiveness as to exclude ignorant credulity, a feature in the development of this subject which has had both ludicrous and obnoxious aspects. It is equally essential, however, that this "prepared mind" be not shut to new ideas and suffer from cynical intolerance. It is, therefore, a singularly appropriate combination to employ the term "prepared-open-mind." A projective imagination is a valuable attribute in visualizing and forecasting relationships between branches of medicine that at first glance may appear to have but a remote association, but unless controlled by a sound conception and acquaintance with the fundamental sciences, is apt to lead to that type of fascinating speculation which emanates from the armchair rather than the laboratory or the bedside.

With this guarded preface, I may perhaps be permitted to launch into the complex subject that has been assigned to me. It may be attacked from several points of view. One may approach it from the standpoint of the patient—that is to say, in what diseases of the ductless glands do symptoms occur of a character which impels the patient to seek the aid of the ophthalmologist, the otologist, rhinologist or the laryngologist; or, second, from the standpoint of the specialist himself, who might well ask whether it is reasonable to suspect an endocrine origin for certain diseases of the eye, ear, nose and throat, whose etiology is as yet uncertain; or, third, from the standpoint of the internist or endocrinologist, who might be interested in pointing out those well recognized endocrine diseases which produce visible or otherwise readily detectible physical abnormalities which the eye, ear, nose and throat specialist could reasonably be expected to recognize in the course of his routine examinations of the fully dressed individual.

The second of these topics, in which you gentlemen are probably more immediately interested, concerns such conditions as cataract, glaucoma, retinitis pigmentosa, keratoconus, retinal hemorrhages and detachment of the retina, vasomotor rhinitis and hay fever, and otosclerosis. In all of these conditions the fundamental cause remains obscure, even though the immediate or exciting cause may at times be recognizable.

In all of them an incretory origin has been suspected, but in none of them has it been definitely established. It is not yet clear whether any relationship at all exists betwen endocrine disease and these important disturbances or malformations; or whether, if a relationship exists, it be direct in the sense of being due to an actual deficiency, or excess, of secretion from one or another ductless gland; or whether the relationship is more indirect; either through fundamental constitutional factors in which imbalance of the endocrine system plays a part; or, whether the mechanism of cause and effect be through the intermediation of the autonomic nervous system. As Ziegler of Philadelphia once phrased it, concerning the last alternative, "The relation between the endocrine glands is close, for, like a pearl necklace, they are all strung on one cord, that of the sympathetic nervous distribution," and one might add that the ramifications of the sympathetic nervous system are particularly noteworthy as they affect the eye, ear, nose and throat. I feel that any consideration at this time of any one of the conditions enumerated above would, at least from the standpoint of the endocrinologist, be a somewhat hazardous undertaking. Clinical observations and clinical deductions, however interesting, have only paved the way for elaborate metabolic studies which must follow in order that the true links, if there be any, may be revealed between such conditions as glaucoma, cataract, otosclerosis, etc.—and the ductless glandular apparatus. Furthermore, a therapeutic solution will only be possible in any complete and adequate sense when more reliable glandular preparations of the character of thyroid extract, insulin and parathormone, are available. It is unfair to conclude that no relationship exists between a particular malady and endocrine dysfunction if extracts have been used whose efficacy is in question. It would be akin to condemning an incretory origin for diabetes (prior to the discovery of Banting's insulin) because of the failure to render the urine sugar free with pancreatic extracts; or to deny a parathyroid origin for tetany because preparations of parathyroid extract (prior to the discovery of Collip's parathormone) were futile in relieving tetanic spasms.

Furthermore, it should be emphasized that, in attempting to appraise the significance of glandular extracts in the treat-

ment of such conditions as glaucoma or vasomotor rhinitis, one must bear in mind that endocrine products have pharmacodynamic powers apart from their rôle as substitutes for specific glandular deficiency. For example, Coates of Philadelphia at one time speculated, in the case of vasomotor rhinitis hay fever and asthma, as to why one person should be sensitized to certain proteins and others not, and postulated the conception that an endocrine constitutional factor might be at the basis of this difference in reaction. I have also fancied that such might be the case, because one has not answered the fundamental reason for an attack of asthma or hay fever by finding that a person is sensitized to a specific protein. Something must underly that person's sensitivity, and whether this is an endocrine anomaly or not is not clear at present. But I cannot agree with Coates when he fortifies his theory with the observation that adrenalin cuts short an attack of asthma and that he conceives, as a consequence of this, a temporary adrenal insufficiency as the cause of the asthmatic attack. An injection of adrenalin results also in reducing intraocular tension in glaucoma and is, in this respect, somewhat similar to its potency in angioneurotic edema, in giant urticaria or in the urticarias of serum sickness where swellings are made to vanish with amazing rapidity. We are by no means justified in concluding that we are dealing here with an adrenal insufficiency in the endocrine sense of this term. This therapeutic effect alone should not impel us to leap to the assumption that glaucoma, or, for that matter, asthma, hay fever or angioneurotic edema, are ductless gland diseases. We could as well imagine a hypopituitarism during labor because injections of pituitrin hasten expulsion of the fetus; and, pursuing this comparison further to its ludicrous but logical conclusion, Hoskins merrily observed that one could as well diagnose a condition of hypocascarism because cascara relieves constipation.

It is apparent, therefore, that the etiologic solution of such conditions as cataract, glaucoma, retinitis pigmentosa, keratoconus, blue sclera, vasomotor rhinitis and otosclerosis is beset with difficulties and that our knowledge of their endocrine affiliations is, at present, meager and inadequate. I would not wish to be misconstrued as denying or ridiculing the plausible

and almost seductive theories which attempt to link up these puzzling maladies with endocrine derangements, but I deem it necessary to point out that any sweeping conclusions in respect to such possible relationships are, to say the least, injudicious and premature at this time.

ENDOCRINE DISEASES WHICH AFFECT THE EYE, EAR, NOSE AND THROAT.

I would prefer, therefore, to tread on safer ground and by means of lantern demonstration pass in review some striking examples of endocrine disease, which, either on the one hand present manifestations of glandular dysfunction in the realm of the eye, ear, nose and throat as a part of general bodily changes, or, on the other hand, present no disturbances in this realm, but produce structural alterations in the body as a whole which are so apparent that they can be recognized, to some extent at least, even in the fully dressed individual.

The speaker then showed slides of the following conditions:*

DISEASES OF THE PITUITARY GLAND.

Acromegaly.—Attention was called to the coarsening of the features due to osseous overgrowth and thickening of the subcutaneous tissues, producing a characteristic facies; the heavy supraorbital ridges, thickening and enlargement of the end of the nose, prominence of the malar eminences, heaviness, squareness and protrusion of the lower jaw, spacing of the teeth; all of which were plainly visible in pictures shown. Attention was also called to the narrowing of the external auditory canal which may occur in this disease and which may result in impairment of hearing; also to the enlargement of the larynx, which is a part of the general tissue hypertrophy consequent to postadolescent hyperpituitarism. Pictures were then shown of perimetric fields in which there occurred a characteristic bitemporal hemianopsia due to pressure of the enlarged hypophysis upon the optic chiasm, it being pointed out that this was a pressure phenomenon, whereas the other abnormalities which had been displayed were hormonic disturbances.

*The pictures referred to below, and many others, are to be found in the chapters by the writer in Blumer's System of Bedside Diagnosis, Section on Diseases of the Ductless Glands.

The speaker showed photographs of an acromegalic, in whom the glandular manifestations of the disease were glaringly conspicuous, and then proceeded to flash on the screen his perimetric fields which, over a period of three years, had been perfectly normal; his hypophyseal tumor had not encroached upon the optic chiasm. The optic atrophy, ocular muscle palsies and disturbances in the sensations of taste and smell were also mentioned as symptoms consequent to pressure upon the pituitary neighborhood.

A picture demonstrating the characteristic spade hand with enormous pawlike widening of the wrists and fingers was shown as a phenomenon of the disease which was not in the domain of the eye, ear, nose and throat, but which could be readily noticed by specialists.

Pictures were shown illustrating the cervicodorsal kyphosis that develops in this disease. The stooped posture, together with the bulging of the sternum and protrusion of the jaw, is sometimes so extreme that the chin comes to rest upon the chest.

A case of acromegaly was shown with pronounced exophthalmos, and it was pointed out that the exophthalmos in this case was due to increased intracranial pressure, probably via the cavernous sinus, but that, because of its outspoken character and the coincidental presence of a small goiter, the diagnosis had been confused with exophthalmic goiter.

Gigantism.—Some pictures were shown of giants, and their glandular implications alluded to.

Hypophyseal Infantilism.—Some pictures of dwarfism due to preadolescent pituitary deficiency were shown, in which no abnormalities of the eye, ear, nose and throat were especially noteworthy except, perhaps, for high arched narrow palate, together with crowding and overlapping of the teeth; but attention was called to the fact that the specialist could readily notice skeletal undergrowth, even in the dressed individual, and realize that it might be due to pituitary deficiency; and there was no law which prohibited him from disrobing the individual in order that he might notice the genital infantilism which characterizes this form of dwarfism.

Postadolescent Hypopituitarism.—Slides were shown illustrating the characteristic girdle adiposity of these individuals,

and reference was made to the sleepiness and headaches which might cause such patients to seek the ophthalmologist for errors of refraction.

Lawrence-Moon-Biedl Syndrome.—Pictures were shown of this rare condition in which the dystrophia adiposogenitalis is associated with mental retardation and retinitis pigmentosa and which affects, as a rule, several members of a family.

Christian's Syndrome.—Slides were shown of this rare condition, in which there is a combination of exophthalmos, membranous bones and diabetes insipidus. In this extraordinary disease the exophthalmos results from faulting of the frontal bone. The skull may approach the consistency of jelly.

DISEASES OF THE PARATHYROIDS.

Tetany.—Slides were shown illustrating the typical accoucheur's hand and also the trophic disturbances in the teeth, consisting of punctate enamel defects, and reference was made to the occurrence of juvenile cataract in tetany and the experimental production of cataract in rats by removal of the parathyroid glands.

DISEASES OF THE ADRENALS.

Addison's Disease.—No pictures were shown of this condition, but attention was called to the bronze pigmentation characteristic of this disease, which is sometimes noted first on the gums, tongue and buccal mucous membranes. The specialist in his routine inspection of the mouth might be the first to discover such pigmented areas and to appreciate their significance.

Virilismus.—Pictures were shown of a remarkable case of adrenal cortex tumor in a woman, 37 years of age, who had not menstruated for the preceding ten months and who, since two years, had developed an appalling hypertrichosis. This extraordinary growth of hair was very marked over the entire body, with heterosexual distribution, and was especially striking on the face, upper lip, chin and neck, where it consisted of a veritable beard. The speaker, however, warned against suspecting adrenal pathology in every woman who was the unfortunate possessor of a few chin whiskers or a faint mustache.

DISEASES OF THE GONADS.

Allusion was made to those peculiar attacks of transient iritis with hypopyon which recur regularly with menstruation; to the cataracts of pregnancy; to vicarious menstruation from the nose, or monthly hemorrhages into the eye, all of which illustrate relationships between the ovary and the eye, ear, nose and throat. Consideration was also given to the question of whether arcus senilis, lenticular opacities and diffuse cataract, when they appear between the ages of 35 and 50, could be considered as evidence of premature senility consequent to a eunuchoid condition.

Pictures were then shown of eunuchoid individuals, and attention directed to the fat pads at the outer corners of the lids which, in part, produce the sleepy lethargic appearance so typical of castrated individuals.

DISEASES OF THE THYROID.

Goiters.—Pictures were shown which demonstrated the well known glistening, staring appearance of the eyes, with widened palpebral apertures and exophthalmos, characteristic of the hyperplastic form of goiter; and pictures of adenomatous goiters with intrathoracic extensions were also shown, in order to demonstrate the resulting pressure phenomena recognizable by venous engorgement of the vessels of the chest and neck. Allusion was made to such abnormalities as inequality of the pupils and such symptoms as cough, hoarseness and dysphagia which might result from the pressure of a goiter on the recurrent laryngeal nerve, sympathetic nerves and cervical veins.

Childhood Myxedema.—Cases of childhood myxedema were shown which demonstrated the typical facies of such hypothyroid children, attention being directed chiefly to the narrow eye slits, stupid appearance, saddle nose, thick, puffy lips and large juicy tongue. The dwarfism that obtains in this glandular deficiency was likewise remarked upon.

Endemic cretinism was mentioned, and the fact that it was probably a different disease from childhood myxedema, and attention was called to the high percentage of deafmutism in regions where cretinism is endemic. A peculiar disease, mon-

golism, was then referred to and its possible endocrine origin discussed. Pictures were shown of the typical slanting almond shaped eyes.

Adult Myxedema.—Cases of adult myxedema were shown. The myxedematous infiltration of the tissues of the face was pointed out, and the dullness and sleepiness of such individuals commented upon. The brassy, low pitched tenor of the voice was alluded to, resulting, as it does, from myxedematous thickening of the laryngeal structures; and the marked impairment of hearing in these individuals was also referred to. Reference was made to the fact that this impairment of hearing was probably due to several factors, such as myxedematous thickening of the eustachian tubes or changes in the auditory nerve itself, and that this was recognizable in the early stages by an increasing inability to appreciate high tones. The characteristic apple blossom cheeks of myxedema were also referred to.

CONCLUSIONS.

It should be evident from the foregoing, and surely would be if the reader could have seen the pictures above referred to, that diseases of the endocrine glands, in their widespread derangement of metabolism and bodily structure, produce oftentimes, as part of their manifestations, disturbances in the domain of the eye, ear, nose and throat. Sometimes these abnormalities will be sufficiently serious to induce the patient to seek the specialist without realizing that the symptoms in his eyes or throat are due to profound disturbances originating elsewhere in the body, and it will then be the province of the specialist to recognize this fact and prescribe the appropriate therapy or refer the patient to an internist qualified in endocrine diagnosis. The procedure is of course the same as when the ophthalmologist advises a general investigation because of retinal abnormalities such as are seen in nephritis, arteriosclerosis and diabetes, or because of choked disc from brain tumors; or when he refers the patient to an internist for the detection of foci of infection that might be responsible for an iridocyclitis or episcleritis.

Finally, it should be stated that ductless gland disturbances are far more common than is generally supposed; that even

the outspoken and extreme grades of disease displayed in the pictures above referred to are by no means rare, and that a thorough acquaintance with the full blown manifestations enable the acute observer to detect and recognize the earlier and less striking signs in minor derangements of endocrine function. It is hoped that this hurried aeroplane view of endocrinology will stimulate a greater interest upon the part of oto-ophthalmologists in this important field of medicine, with a realization of its difficulties and complexities, and an appreciation also of its brilliant achievements. It is hoped that such an understanding will encourage a more intensive cooperation between the oto-ophthalmologist and the internist, both in the recognition of endocrine disorders and in the attitude, previously referred to, of the "prepared-open-mind" toward future investigations.

It is, however, desirable that future contributors to this field strive to separate fact from fancy. It would be helpful to the average uninitiated reader if such writers stated frankly which of their conclusions were derived from bedside observation or laboratory experiment, and which of their conclusions emanated from imaginative cogitation. Unfortunately, in the past, many writers have used the same brush of conviction in painting alike their clinical or laboratory data and their arm-chair speculations. Theorizing in endocrinology is not a crime, so long as it is properly labeled. A penetrating and prophetic vision is a rare gift, but it must not parade under the banner of verified truth.

LIX.

COMMENTS AND SUGGESTIONS ON OPERATIONS ON THE MAXILLARY AND FRONTAL SINUSES.

By T. E. Oertel, M. D.,

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Someone has said "An operation is a confession of inability." Doubtless this is true. The more we learn, the less becomes the necessity for surgical measures. Unfortunately, our knowledge has not yet progressed to the point where the scalpel may be relegated to the museum as a relic of an unenlightened age. So long as surgical procedures are necessary, we can only strive to perfect our technic so far as our skill and present knowledge will permit.

The fact that so many operations have been proposed for the relief of chronic sinusitis evidences faults in all of them, for if any were perfect it would not have been necessary to seek to invent improvements in it. We must allow that no operation is as satisfactory as resolution to the normal by other means, but when these are beyond our ken and surgical intervention is the only recourse our next care must be to choose that procedure which will best meet the indications of the case and which at the same time promises to produce the minimum amount of trauma with its subsequent scar formation or other untoward postoperative results.

I think it is not too much to say that chronic suppurations of the frontal sinus present more difficulties than do those of any of the other prenasal cavities. The reason for this is based upon the anatomic peculiarities of the part. The frontal sinus may be likened to a funnel, the small end of which terminates in the infundibulum. The osteum is more or less elongated and narrow. The hiatus semilunaris, through which drainage from the frontal sinus must pass, is also narrow and easily blocked. If the secretions in the frontal sinus become either profuse or thick, ready flow into the nasal chamber is retarded. Swelling of the mucosa lining the channels of exit results from the irritating pus and further obstructs drainage.

Finally, polypoid degeneration or granulations may ensue and entirely block the passage. Upon the seriousness of such a condition it is not necessary for me to comment.

All too often it is not until this stage has been reached that the patient consults a rhinologist. Until now he has suffered at the hands of some friendly druggist who has given him free the benefit of his wide experience in the treatment of "catarrh," and incidentally sold him one of the nostrums from the shelves, or his family physician has hastened to his relief with aspirin in one hand and argyrol in the other.

Negative pressure coupled with suitable medicaments may still be productive of good results. All to often we must turn to some form of operative procedure with the hope of a successful issue. Which shall it be, intranasal or extranasal? Which of all the numerous methods advocated by textbook and monograph best fits the case in hand?

It is, for myself at least, a difficult matter to decide. Intranasal methods are often satisfactory, often disappointing. The type of nose has much to do with the decision. Unfortunately it is in the narrow nose that sinus infections most often occur. Manifestly this is due to improper aeration and drainage. Often a high deflection of the nasal septum obstructs both view and access to the middle fossa on the affected side. In such a case it is necessary to first do a submucous resection of the septum before it becomes possible to reach the ethmoid region. Even when this has been done we may have only a narrow slit through which to work.

I am free to confess that exenteration of the ethmoid capsule is to myself not a small procedure. The variable anatomy of the part, together with the surrounding zones of danger, its inaccessibility, the blood covered field, render it a measure of extreme difficulty. Nor have I often been able to assure myself that all of the numerous cells have been well opened.

When it is necessary to do a submucous resection of the septum for the purpose of gaining access to the middle fossa shall it be a two stage operation or be done coincident with the further and more serious procedures? If done as a two stage measure, we must wait for the septum to heal before doing anything more. On the other hand, we run the danger of infection of the septal mucosa by the secretions of the in-

fected sinuses above and the more so on account of added irritation from the necessary extensive manipulations.

Often we must be influenced by the circumstances of the patient. His financial condition may preclude any long hospitalization. His business interests may demand that form of operation which will promise a minimum loss of time.

Are we ever justified in doing the disfiguring Riedel operation which leaves the patient with a flat, bullfrog face? I have done it in two instances. Both of the subjects were women well advanced in life. Both were poor. They came from the country to have something done which would relieve the pain and effect a cure in the shortest possible time. I explained the situation at length. With the Riedel operation I could promise a cure. With no other operation could I say as much. Disfigurement would be the price they would have to pay. They chose to pay it. What might I have done better?

It is easy to ask questions. Much easier than to answer them. My purpose in doing so is to bring about a free discussion of the subject, with a hope that each of us may benefit and the surety that I shall learn much which will profit myself and my patients of the future.

Perhaps it is not too bold an assertion to say that operations in chronic infections of the maxillary antrum are more uniformly productive of good results than those upon other of the accessory sinuses of the nose. Many reasons may be adduced for this. Chief among them may be mentioned accessibility of the operative field, freedom from extensive tissue destruction in the nasal chamber and permanent drainage. I am, of course, speaking only of those more radical operations which we employ in chronic conditions with polypoid degeneration of the antral mucosa.

Doubtless we have all run the gamut seeking for the best method to adopt as a standard routine measure and failing to find in any the fulfillment of our hopes. In other words, each has both advantages and disadvantages, some of which it may be well for us to consider.

The Caldwell-Luc provides easy accessibility to the antrum, except at the anteromesial angle, which is around the corner and out of the reach of any but an angular instrument. It is also somewhat difficult to make satisfactory drainage into the

floor of the nasal chamber, because the bony wall under the lower turbinate is not easily accessible. In displacing the soft tissues upward in order to expose the canine fossa, often too much pressure is brought to bear upon the superior maxillary branch of the fifth nerve at its exit from the bone, and unpleasant results follow. Numbness and often apical abscesses of the teeth ensue on account of interference with their nerve supply or nutrition from section of the bone directly above them.

To obviate continued infection from diseased mucous membrane or bone in the pyriform angle, Denker proposed to extend the Caldwell-Luc resection of the bone through this angle and continued the resection along the mesial border under the lower turbinate in order that a flap of mucous membrane might be turned into the floor of the antrum to guard against closure of the opening by granulation tissue.

The objections of the Caldwell-Luc operation obtain in greater degree in that advised by Denker, on account of the more extensive destruction of the anterior bony wall, as well as the longer incision in the soft parts necessary for the proper exposure of the bone.

To obviate these objections, Canfield devised his method of attacking the pyriform angle by an incision just within the vestibule, elevating the periosteum and mucous membrane under the turbinate, doing a submucous resection of the inferior turbinate bone and turning into the floor of the antrum a flap of mucous membrane as in the Denker operation. The two outstanding objections to this operation are the destruction of the turbinate and the limited field of operation. While it is possible to inspect the antrum through a small speculum, it is quite difficult to thoroughly clean out diseased mucous membrane or polypi with any degree of certainty and then only by the sense of touch, as the instrument employed fills up the opening and obstructs vision.

Skillern[1] advocates a modification of this operation in that he does not resect the turbinate. He uses a pharyngoscope to inspect the roof of the antrum and an ear speculum for farther observation. To my mind, the restricted opening through which one must work is a distinct handicap. Through such a small hole one can neither see nor work with facility, nor can

instrumentation be surely carried out. I think it is axiomatic that it is always desirable to have the field of operation under direct vision.

Marquis[2] regards the Denker operation as superior to the Canfield for the reason that the Denker operation allows greater retraction of the soft parts. He points out the fact that the Denker operation has the objections of causing loss of sensation in the lateral incisors and bicuspid together with numbness of the lip and cheek, and considers this an objection to an otherwise ideal mode of procedure.

Sauer[3] says: "Personally I have not done the Denker operation in recent years, as the same result can be obtained with the Caldwell-Luc, and the reaction in the latter is decidedly less. . . . Anesthesia of the face as a complication following frontal and maxillary sinus operation has to do with a condition that is chiefly important only as an accident of some inconvenience to the patient but of no great import. . . . Most cases reported seem to clear up eventually, even though the nerve involved is usually severed during operation."

Theobald[4] advocates an intranasal modification of the Canfield procedure, much like the "preturbinal" operation of Skillern.

Ivy[5] maintains that little or no permanent damage is done to either the blood or nerve supply of the teeth when the anterior wall of the superior maxillary bone is cut away, as in the Caldwell-Luc and Denker operations. He thinks "Nothing is so satisfactory for exact diagnosis and treatment as exposure of the interior of the sinus to direct inspection and palpation through a large opening from the mouth through the buccal wall."

In considering possible damage to the blood or nerve supply of the teeth in cases where such operations had been done, he says: "Practically all patients noticed a numbness in the teeth and gums beneath the buccal incision, which lasted only a few weeks or months in some, in others for a year or more. He did not find any evidence of pulp degeneration or loss of vitality in the teeth of the twenty patients examined.

Allowing, for sake of argument, that no serious damage to the teeth results from destruction of the wall of the bone above them, we must all admit that it were better if destruction of this

region could be avoided and at the same time a clear field of operation be obtained such as the open operations provide.

This reasoning caused me to try a procedure which I believe is of value and submit for your consideration. I do not know that I can presume to call it a new operation, but I have not been able to find that anyone has previously employed this technic.

An incision under the lip is made, as in the Caldwell-Luc and Denker operations. Care should be taken that it is not too low. I think it is best made at a point near the fold of the lip, and it should extend from the midline laterally for about an inch or perhaps a trifle more than this. The cut should extend through the soft parts and periosteum. A periosteal elevator is now employed to strip up the soft parts until the apertura pyriformis is well exposed. An angular elevator is now used to strip up the mucous membrane under the lower turbinate from its insertion well down on the floor of the nose and for an inch or more backward, or so far as is deemed advisable, to make a flap of mucous membrane to be turned into the floor of the antrum at the close of the operation. I have not found it necessary to remove the tip of the turbinate or resect this bone, as Canfield recommends. A cutting forceps is now applied to the pyriform crest and a sufficient amount of the anterior angle removed to open the antrum and allow the forceps to be turned along the mesial wall and cut out the bone from the attachment of the turbinate well down to the floor of the nose and as far back as it is desired to make the permanent opening into the nasal chamber. It is well now to cut the flap of mucous membrane and turn it backward into the nose until further manipulations are completed. The antrum is now exposed so that it is possible to directly inspect its whole extent. There may be difficulty in seeing portions of the floor above the roots of the teeth with sufficient clearness, in which case I use a small laryngeal mirror, previously heated by dipping into hot water. This often proves of great service, and it seems to me is better than the pharyngoscope on account of its simplicity.

The antrum can now be cleaned out, as the case may demand, and the nasal flap is turned down into the antral floor. The antrum is lightly packed with a gauze wick, the end of

which is brought out of the nose, thus holding in place the turned in flap. The wound in the buccal mucosa is closed by two catgut sutures.

The special advantages of this method are:

1. Ease of accessibility to the antrum.
2. A minimum of destruction of the anterior wall.
3. The possibility of direct inspection of nearly the entire cavity—all of it by the aid of the mirror.
4. Greater freedom from numbness and tenderness in the anterior teeth on account of disturbance of innervation.

It may be argued that it is well to avoid a route through the mouth on account of the extensive cut necessary, but I believe this is no great disadvantage, as it readily heals, usually within a day or two, and seems to give little discomfort. There is nothing like the same degree of reaction that either the Denker or Caldwell-Luc operations produce.

You will recognize that I have endeavored to combine the good points in the two types of operation, the intranasal and extranasal, and at the same time to do away with some of the things that are objectionable in other operations. How far my efforts have met with success will be determined by the test of time.

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LX.

LARYNGEAL CYSTS—REPORT OF CASES.

By W. B. Mason, M. D.,

Washington.

I am presenting this paper to call attention to the comparative rarity of these tumors and to the very meager literature on the subject—in fact, the paper is a brief summary of the literature that I have been able to review; and while I have probably fallen far short of reviewing all the literature, I have made quite a careful search, and believe, judging from the sameness in all cases reported, that I have pretty thoroughly covered the salient points of the disease. When my first case came under my observation and I was finally able to distinctly see the tumor, I must confess that I was very much puzzled. But after studying the characteristics of the growth, I decided that it could be nothing but a cyst. It then occurred to me that I didn't know very much about cysts of the larynx; in fact, I could not recall ever reading anything about them, though it did seem to me that I had seen the term in print somewhere. Consulting the only textbooks I had at hand at the time, I found no mention of the subject. Later, I investigated a number of textbooks; many of them did not mention the subject, and those that did devoted from one paragraph to half a page to it. This is probably explained by the fact that the disease is not only quite rare, but is scarcely ever serious. In regard to the rarity, my experience leads me to believe that in many instances it is very easy to overlook these tumors.

The patient is nearly always a nervous old person, who has already diagnosed the case as cancer in the worst form, and the extremely movable growth flapping around in one's larynx with every respiratory effort and act of deglutition does not tend to allay nervousness or make our already difficult examination easier.

One of my patients reported that she had been in the hands of an experienced laryngologist for six weeks prior to her

consulting me, without his having discovered the cysts. If the growth is small, has a short pedicle and occurs very late in life, as they usually do, it may give rise to practically no symptoms and never be suspected. In view of these facts, it is quite possible that laryngeal cysts may be far more common than is generally supposed. In my review of the literature I found the average estimate of the proportion of laryngeal cysts to other tumors of the larynx to be from 2 to 5 per cent. One man only went above 5 per cent, and he estimated that 25 per cent of all the tumors of the larynx were cystic. I think that most of you will agree with me that this is absurdly high. In one hundred cases of growths of the larynx published by Sir Morrell McKenzie only two were cystic. It would be impossible to estimate with any degree of accuracy the relative frequency of the occurrence of cystic tumors of the larynx, because there are probably many laryngologists who have seen hundreds of laryngeal growths without ever having seen one that was cystic.

Laryngeal cysts differ in no particular from cysts of any other region except in size. The pathology is the same as that of a mucous retention cyst elsewhere, viz., an acute inflammation causing stoppage of the duct of a mucus gland, and this process increasing with each subsequent inflammation until there is sufficient accumulation to cause the cyst.

As already stated, they rarely occur in any but very old people. I found one case, reported by Dr. Cassellberry (Jour. A. M. A., 1891), of the occurrence of a cyst of the arytenoid in a woman, 25 years old, but found no other cases reported in patients under 60 years of age.

There are generally found on the epiglottis, especially on the lingual surface, about the glossoepiglottic ligaments, are occasionally seen in other regions of the larynx, and on the laryngeal surface of the epiglottis, on the aryepiglottic fold, and rarely on the arytenoids. (Dr. Cassellberry's case was the only one I could find reported as situated on the arytenoids.) Several writers make the statement that they are practically always situated anterior to the middle third of the vocal cords.

They are smooth, rounded, gray in color, semitranslucent in appearance and vary in size from a pea to a pigeon's egg.

Small vessels are seen coursing over the surface of the tumor, producing a very characteristic appearance. On puncture they are found to contain a mucus fluid, although suppuration within the cyst is not uncommon.

They give rise to very few symptoms, there is never any pain, and the sensation of having a foreign body in the throat seems to be the most prominent symptom. In my case this was practically the only subjective symptom. (In localizing the foreign body, one of my patients invariably pointed to the region of the tonsil on the affected side, which was, of course, misleading.) Desire to clear the throat is a fairly constant symptom; one of my patients was considerably troubled by it, while the other was not.

Dysphagia is purely mechanical and is only present after the growth attains considerable size. There is no dyspnea until the cyst becomes quite large; then it may be a very troublesome symptom, especially if the pedicle is long and the cyst is very freely movable. In this case, inspiration may draw the cyst down into the larynx, thereby seriously interfering with breathing. Cough is rarely ever a marked symptom.

The only difficulty in making a diagnosis is in being able to get a good view of the larynx. The appearance of a cyst is so entirely different from that of any other tumor of the pharynx, with the possible exception of pemphigus, that the diagnosis is perfectly simple if your patient allows you to get a good view of the larynx. However, this just where the difficulty lies, because these patients are nearly always nervous old people, in whom a laryngeal examination is hard under any circumstances, and doubly so because of their fear that they have a cancer. In addition to this, if the cyst is of moderate size and has a pedicle of the right length, it is surprising how often it can hide between the anterior surface of the epiglottis and the base of the tongue. You may have to make repeated examinations in order to get a glimpse of it. In such a case it is not surprising that the cyst is not detected, and may be more or less an accident that it is.

Pemphigus might be mistaken for a cyst. The most prominent symptom of pemphigus is pain, a symptom never present in cases of cyst of the larynx. Pemphigus appears as a few clear, transparent blebs, which in a few hours become turbid,

then yellow; finally they burst, discharge and collapse, leaving the appearance of a false membrane, which will suggest the probability of diphtheria. When these blebs become turbid they are not unlike a cyst, particularly if the latter is not pedunculated. However, close observation of a case would easily differentiate between pemphigus and a cyst.

Laryngeal cysts are rarely, if ever, serious unless entirely neglected and allowed to reach a very large size; in which case they may become a serious impediment to speech, respiration and deglutition. The consensus of opinion is that they form very slowly.

There is no danger attending the operation, and it is most easily done under local anesthesia; in fact, there is so little sensation in the cyst that cocain is necessary only to enable you to make a better examination and prevent gagging.

There is no tendency to hemorrhage and the effect on the voice is nil, except in a case that has been neglected, as in a case reported by Dr. G. H. McKenzie (Lancet, 1895), in which he described the cyst as a large pear shaped body, lying on and obscuring the left vocal cord and a part of the left ventricular band. It was slightly movable and was attached by a short pedicle near the anterior commissure of the vocal cords. Removal gave immediate and permanent relief, but disclosed the fact that the left vocal cord and a part of the left ventricular band had completely disappeared from pressure and erosion.

The prognosis is good, and, provided you are able to make a good permanent opening in the cyst wall, the tendency to recur is very slight. Simple incision which will evacuate the cyst, as well as any other procedure, is not sufficient, because the fluid will surely reaccumulate. Removal or destruction of a considerable portion of the cyst wall must be resorted to. If the cyst is on the lingual surface of the epiglottis, pedunculated and freely movable, it is sometimes possible to depress the tongue hard, seize the cyst, pull forward and remove a large part of it with curved scissors. If it is a sessile growth, this will not be possible, and a laryngeal punch or galvanocautery will best enable you to make a permanent opening in the wall of the cyst. The cautery is neither practical nor safe in the deepest parts of the larynx. The punch or a cutting forceps like McKenzie's will afford the best means of dealing with these

cases. It is not at all necessary to remove the entire cyst wall. A good permanent opening will prevent reaccumulation, the cyst will collapse and all traces of it disappear in a few days.

Case 1.—Mr. W., aged 70, consulted me first on November 7, 1912. Complained of "frog in throat" for past few days. On examination, I found that he had a mild pharyngitis. No inflammation of the larynx whatever. There was some mucus on the vocal cords, which an alkalin spray removed immediately. At that time a laryngeal examination was very difficult and a good view of the larynx was impossible. I treated the pharyngitis along the usual lines and saw him four times in the following two weeks. After the second treatment the pharyngitis had entirely cleared up, and then he complained that while his throat felt better there was something wrong, and he felt as if there was something in his throat that did not belong there. He was getting more nervous every time I saw him, clearing his throat incessantly and asking me at each visit if I were sure he did not have cancer.

Finally, by exercising the greatest patience, I succeeded, with the aid of a 10 per cent cocain spray, in getting a satisfactory view of his larynx, and at last there suddenly popped into view this peculiar looking growth, smooth, rounded, gray in color, semitranslucent, and about the size of a small cherry. Minute vessels could be distinctly seen coursing over its surface. It was freely movable and frequently disappeared from view during examination. Judging from its movements, it was evidently attached by a pedicle, but just where it was impossible to say.

After getting a good look at it, I was sure that it could be nothing but a cyst, and the temptation to incise it and relieve the old gentleman was very great. However, I decided to have someone else see the case before operating. I arranged a consultation with Dr. J. H. Bryan at once, who seemed to think the treatment I suggested, viz., to punch a large hole in the cyst, was proper and would effect a permanent cure.

This I did immediately and without the least pain to the patient. He simply coughed, spat up the contents of the cyst, which was pure mucus, and felt relieved at once, so much so that from that time on satisfactory examination was perfectly

easy. When the cyst collapsed I could see that it was attached along the glossoepiglottic ligaments. Three days later no trace of the growth could be found. I kept him under observation for about three weeks, during which time there was no sign of recurrence. I heard from him six months later that he was perfectly well.

This patient lived eleven years in perfect health, leading quite an active life, having no recurrence of the laryngeal trouble. Died during 1923 following an attack of influenza.

Case 2.—Mrs. B., aged 72, consulted me first on August 29, 1913, and gave the following history: "Following an attack of grippe last winter my throat became irritated and has continued so ever since. There has never been any pain and practically no cough or hoarseness, only the constant sensation that there was something in my throat. About six weeks ago, after a slight cold, this sensation was so much increased that it became very annoying and interfered with my sleep. At that time I consulted a throat specialist, who has been treating me ever since. My throat has gotten no better, and I have decided to make a change and see if you can help me."

She was extremely nervous, had a very small mouth and throat, and a tongue that you could not pull forward more than a quarter of an inch. I have never seen her equal for gagging, and when she did, the result was always the same, namely, a complete closure of the throat, half an inch above the epiglottis. Finally, after many applications, I almost completely anesthetized her pharynx, and she was able to hold her throat open for very short spaces, giving me an occasional glimpse of the larynx, which showed no sign of inflammation.

After repeated examinations, a small growth suddenly appeared alongside the left border of the epiglottis, as if it had just been squeezed out from between the epiglottis and the base of the tongue. It was very similar to the growth found in case No. 1, only it was about one-third smaller, a little less movable and very much more yellow in appearance.

I made several attempts to seize it with a punch and cut a hole in it, but was unsuccessful on account of the fact that I had no instrument with a short enough curve for her throat. This, of course, made her gag all the more. Finally, in desperation, I took a small laryngeal knife, and, by drawing

it from below upwards, succeeded in splitting the cyst wide open, from bottom to top, evacuating a small quantity of very yellow mucopurulent material.

This gave her considerable relief at once, and to some extent facilitated laryngeal examination. Her throat and tongue were so very sore from prolonged manipulation that I only attempted to spray her throat with alkalin sprays during the next few days. I then went away on my vacation.

On my return on October 1st, she came in again, saying there was trouble there, because she still had some of the same sensation of a foreign body in her throat. At this time I was able to make a more satisfactory examination, and after assuring myself that there was no reaccumulation in the cyst, I looked more carefully for an explanation of her continued trouble, and finally discovered another cyst, a sessile growth on the base of her tongue, back towards the epiglottis.

I made one unsuccessful attempt to get this with the punch —then split it open with the knife. It refilled in about a week, and then I burned away a large part of it with a galvanocautery. This was a very easy procedure and should have been done at first. Her throat was entirely well within a week.

She enjoyed excellent health for nine years, with no recurrence of this trouble. Died of pneumonia in 1922.

1738 M Street.

LXI.

ON THE USE OF ISOTONIC SOLUTIONS FOR NASAL ADMINISTRATION.

By J. E. Rush, M. D.,

Lexington, Ky.

From time to time a small percentage of the students treated for nasal conditions, in the dispensary of the University of Kentucky, have stated that while the adrenalin or cocain used for the primary shrinkage relieved them greatly, much irritation was experienced when the usual antiseptics were subsequently applied. Because of the fact that the dispensary is freely used by our large student body and that those with "colds" (with but very few exceptions) use the dispensary on the first appearance of symptoms, it was felt that this matter warranted investigation. Too, it was thought that its solution might be interesting to others who did not have such a large group to make observations upon.

We had been using argyrol following the cocain administration, but it was determined to substitute solargentum for it, and later still the solargentum was replaced by silver nucleinate. The same observationus were made, namely that certain students experienced discomfort following administration of the silver salt.

It was thought that a decrease in the concentration of the antiseptics used might help toward a solution of the problem, but it was found that changing the concentration of the silver salts (whether argyrol, solargentum or silver nucleinate) from 10 to 5 per cent, and later even to 2 per cent, did not eliminate the suggestions from some students as given above.

Just about this time the writer developed an acute rhinitis which later became chronic and was complicated by a sinusitis. The same treatment was used, namely, shrinking the tissues, followed by one of the antiseptics above named, with the result that the shrinking afforded great relief, but the administration of argyrol, solargentum or silver nucleinate, in addition to being quite irritating, seemed to cause a return of the

original anatomic condition. The tissues seemed to become engorged and turgid following the administration of the silver salt, and the discomfort came on so rapidly that it was thought that it must be due to the treatment. It was present irrespective of the silver salt used or its concentration (within the limits specified above).

It occurred to the writer that the condition was probably due to the use of a hypotonic solution, which, following the shrinking due to the cocain, might, because of osmosis, produce the effect experienced. It was determined to substitute physiologic saline solution for the distilled water used in making up these preparations. The osmotic pressure exerted by the organic silver salts themselves was thought to be negligible, and the physical effect of their addition to physiologic saline was not taken into consideration. It was reasoned that if a hypertonic solution did result from their addition its administration probably would be therapeutically sound.

It was necessary to determine if there would be a precipitation of the silver organic salts on the addition of this quantity of sodium chlorid. Testing all concentrations of the antiseptics used, it was found that no precipitate resulted, even though the solutions were kept for several days.

The subsequent use of isotonic solutions of argyrol, solargentum and silver nucleinate, even in those whom we had come to know had rather sensitive nasal mucous membranes, was very gratifying. Several chronic cases spontaneously remarked on the fact that these solutions were less irritating, and in instances where applications of the isotonic and hypotonic solutions of equal concentrations of the same antiseptic were made to the two nostrils of a given individual, report that the hypotonic solution was distinctly more irritating occurred in every case.

LXII.

ADDRESS DELIVERED BY DR. J. HARDIE NEIL, PRESIDENT OF THE OTOLARYNGOLOGICAL SECTION, NEW ZEALAND MEDICAL CONFERENCE, AT THE OPENING SESSION, AT AUCKLAND, N. Z., MARCH 1, 1924.

Gentlemen—On behalf of the New Zealand Branch of the British Medical Association, I extend a cordial welcome to the visiting members of the profession.

This conference is an outstanding one, because of its international character, and we associate our Australian confreres with us in expressing our gratification at the presence among us of two distinguished American colleagues who have for years been prominent in the ear, nose and throat world.

Dr. Francis P. Emerson of Boston and Dr. Thomas Hubbard of Toledo, Ohio, have been consistent contributors of papers to the journals for many years, and their contributions to the society discussions have been very illuminating and of great value. We must take this opportunity of thanking them for their many additions to the storehouse of knowledge in connection with our specialties. Contributions to the journals are broadcast to an audience limited only by the confines of civilization. The help and comfort they afford to isolated workers in guiding and advising them as to methods tried, approved or rejected in large and well equipped clinics, is of immense benefit to the profession and community. Many of us have been helped and comforted and steadied in difficult and perplexing questions by these contributions from the masters of the specialties.

Our American confreres are particularly frank in discussing results, and have the hallmark of the strong man who will table unsuccessful results and freely discuss the possible methods of improvement.

Their search for knowledge is insatiable, and their numerous well endowed research institutes have fostered the spirit of investigation and progress. Their consistent efforts to improve

operative technic have enriched the whole medical world. On behalf of the Australasian profession we offer our grateful thanks to these gentlemen for their sacrifice in attending as representatives of the American Laryngological Association and the American Otological Society.

We miss among our Australian colleagues Dr. Herbert Marks of Sydney. We much regret the illness which has at the last moment prevented his coming. His absence will be a great loss to us, for he is undoubtedly the President of Australasian presidents.

It is particularly gratifying to have with us Sir Henry Lindo Ferguson. Those of us who have been his pupils realize that he was always solicitous for the welfare of the students, and no one has been more consistent in maintaining the prestige and dignity of the profession. Furthermore, he has steadfastly maintained that the surest way to help the public in need of medical aid, in more or less isolated areas, is to raise the general standard of education. In short, his professional eminence and faithful work for the public and professional good have been rewarded by the King with a decoration, which has met with universal gratification.

Distance only prevented us from being honored by members of the otological and laryngological sections of the Royal Society of Medicine. The ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, the Laryngoscope and the British Journal of Laryngology, to the two former of which our American guests are distinguished contributors, demonstrate the cordial relations which exist among English speaking nations in our specialties.

It is with great pleasure that I call on Dr. Emerson to read the opening paper.

LXIII.

ACUTE SUPPURATIVE OTITIS MEDIA CAUSING SEPTIC ARTHRITIS WITHOUT OBSERVABLE SYMPTOMS OF INTERVENING MASTOIDITIS OR OF INVOLVEMENT OF THE LATERAL SINUS.

By Frederick A. Kiehle, A. B., M. D.,

Portland, Ore.

The picture of a septic process that originated in a simple suppurative otitis media leaping directly into the blood stream and attacking distant structures is unusual and has been all but disregarded in the literature of otology. Recent writers, among them particularly Kopetzky, contend that the mastoid cells participate in all cases of otitis media. This may well be true. And yet it may be possible that at times the invasion of the mastoid cells and the attack upon the lateral sinus wall may not be distinguished by the usual accompanying signs.

In considering the pathology of the extension of any septic otitic process we have to consider one of two routes: First, direct extension, as in meningitis, where the organism, by erosion of the attic or antral roof, has come into immediate contact with the dura. Or, second, the lymphatic route. The rôle of the lymphatics in infective processes has long been accepted, and, generally speaking, the richness of the venous and lymphatic supply of a part governs the ease of exit of infectious material. Hence a bony cavity, such as the tympanic cavity, with but a slender lining of mucosa and with a limited lymphatic supply, offers relatively little opportunity for passing along infection.

We are quite accustomed to the sight of systemic extension of purulent otitis media with the intervening manifestations that ordinarily accompany inflammation of the mastoid cells and penetration of the lateral sinus wall with thrombosis formation following. The two cases here reported raise the query

as to whether or not discernible evidences usually associated with mastoiditis and thrombosis formation may be entirely absent, while yet the infective process is fully under way. The query also arises whether absorption of septic matter from the tympanic cavity could take place through the lymphatic stream direct without the aid of the sinus channel. Where the otitis is bilateral the opportunity is doubled and absorption from this increased area may well have been a factor in the cases to be detailed.

Briefly, the two cases are as follows:

Case 1.—K. C., schoolboy, aged 8. The child had been generally well, except for pertussis at the age of 18 months. Aged 4, he had concurrent abscesses in the two ears, which ruptured and ran a simple course to recovery. Aged 5, measles, accompanied by severe tonsillitis, following which tonsillectomy and adenoidectomy were performed. On January 10, 1923, he was brought to my office with a history of having caught cold three or four days before. He was now complaining of right earache for the past 12 hours. He was taken at once to the Portland Eye, Ear, Nose and Throat Hospital, given gas and oxygen and a right myringotomy performed. The laboratory test of the discharge of this ear showed streptococcus hemolyticus. The next day the drum of the other ear, which on examination the evening before had appeared but slightly congested, broke spontaneously and ran a simple course to recovery. The right ear ran for several weeks, but without pain, edema, glandular enlargement, mastoid swelling or mastoid tenderness, either deep or superficial—in short, nothing that seemed to justify surgical opening of the mastoid cells. The day following the paracentesis an eruption appeared on the chest, somewhat resembling a scarlatinal rash, but it quickly receded and entirely faded within 24 hours. Five days after the paracentesis the fever, which had been ranging between 99 and 101 degrees, took on the character of a septic temperature, mounting from 99 to 105 degrees with a distinct chill. The right hip was swollen and painful on rotation, and there was an area of redness over the left elbow. The blood showed streptococcus hemolyticus. The patient was immediately returned to the hospital in charge of an exceedingly competent orthopedist, who without delay opened and drained both hips.

From there on it was a story of long and protracted convalescence, though no other joints were attacked. The struggle was a hard one, with slowly waning vitality, recouped only when direct transfusions of whole blood were made from the mother (March 10, 250 cc.; March 12, 100 cc.; March 14, 150 cc.; March 22, 90 cc.; March 24, 180 cc.), five in all, with immediate and spectacular convalescence ensuing. Erosion of the acetabular rim has left the boy noticeably lame, but he walks without crutches, and the orthopedist promises greater improvement from an orthoplasty when the patient arrives at maturity.

The ears at no time furnished other symptoms than those noted, and though frequently X-rayed and watched carefully, never gave any further indications of trouble. Air conduction is reduced to approximately two-thirds in each ear. Bone conduction is normal.

Case 2.—F. M., schoolboy, aged 10 years. Early history inconsequential. In June, 1923, he had a left acute otitis media and in October, 1924, a double otitis. In each case a paracentesis tympani had been performed. Healing was prompt. Tonsils and adenoids were removed shortly after the first otitis noted above. March 24, 1925, he was brought to me with a history of having caught a cold a few days previously. The next day both ears began to ache slightly, and by the following day it was necessary to incise both drums. The ears drained freely. The accessory nasal sinuses were throughout negative, and at no time following was there any further complaint as to the ears. At no time were there any symptoms that suggested the advance of the otitic process into the mastoid. However, on April 4th, eight days after the onset of the first ear pain, the right knee and the right elbow began to swell, and the temperature, that had hovered over 99°, rose to 104.2°. The right thigh showed a circumferential measurement of more than two inches greater than the left. The entire leg was tender, but the swelling was most marked over the lower end of the femur. Lymphocyte count 14,200, polymorphonuclears 90 per cent, small lymphocytes 8 per cent, large 2 per cent. The blood gave positive streptococcus hemolyticus findings. Under the preoperative diagnosis of osteomyelitis of the right femur, the patient was prepared for operation, again by

an exceedingly competent orthopedist. Pus exuded on dividing the fascia, but the femur was drilled in four places without yielding pus. Exploration of pus pockets between the fascial sheaths, however, revealed the extension from the knee joint proper, indicating that the capsule had ruptured and that the pus under pressure had followed the sheaths of the muscles into the thigh. Several ounces of pus were evacuated from the knee joints from incisions on both sides of the patella. The swelling on the right elbow proved to be a superficial abscess. A general septicemia developed and death ensued in five days after the operation, or exactly eighteen days from the initial earache. No opportunity was given for a postmortem examination of the condition of the lateral sinus walls.

Such are the histories, greatly abbreviated, presenting only the important points and omitting much detail.

To summarize: Two lads of an age when infections travel rapidly develop a double otitis media, which in each instance progresses along the usual course. In neither was there anywhere in the body other discernible foci to account for the general infection that arose. In both the blood showed the presence of the same organism (streptococcus hemolyticus) that had been discovered in the aural discharge. But tenderness, both superficial and deep, both over the antrum and the tip, was absent. There was never the slightest indication of redness or of edema, none of the "feeling of the ear" common in children, no bulging of the posterosuperior meatal wall. The usual moderate temperature and serosanguinous discharge, changing to purulent in a few days, alone associated themselves with an otitis. The temperature took on a septic type with the manifestations of the arthritis, and no other symptom significant in any way of sinus thrombosis was present—no tautness of cervical muscles, no tender or enlarged cervical glands.

The literature gives many articles on focal infection leading to arthritis, particularly with tonsils, teeth and sinuses as the source, but invariably the ears are passed over very lightly as a possible factor in this condition.

L. Grünwald, writing on "Otitis and Osteomyelitis" in Der Zeitschrift für Nasen u. Ohrenhlk., questions whether the osteomyelitis does not cause the otitis rather than vice versa.

In all cases he cites, however, there existed mastoid tenderness and sinus involvement.

Oppenheimer (N. Y. Journal, July 8, 1916) quotes a similar case of inflammatory processes in joints secondary to suppurative otitis media, and mentions two of Wolf. Wolf, however, felt that in his cases the otitis was secondary to the arthritis.

In both cases detailed above the otitis was "post hoc" and apparently also "propter hoc." In both of these cases the ears were repeatedly watched in the hope that they might furnish grounds for interference. Furthermore, in both these cases the accessory nasal sinuses and the throat were eliminated as possible factors. Both were repeatedly examined by consultants in unavailing search for other foci of infection that might have been productive of the arthritis.

We may, I believe, in both cases postulate a wide open aditus on each side, a lateral sinus lying far forward and susceptible to easy attack through large contiguous mastoid cells. Then a surreptitious entrance of the infected organisms into the blood stream via the lateral sinus without formation of a clot, or at least with nothing more than a minute mural thrombus, in the formation of which the phenomena of the invasion of the sinus, viz., chill and septic temperature, corresponded with the appearance of the septic symptoms of the arthritis. In other words, the attack upon the joints was practically synchronous with the invasion of the lateral sinus.

In the presence of such a picture, would one have been justified in laying bare a lateral sinus, in the one case within five days and the other within eight days of the initial earache? With the courage gained from combating with questionable success the one case and with distinct failure the other, I believe that under like circumstances the condition of the lateral sinus should certainly be investigated. In the presence of a double otitis, each ear apparently equally inoffensive, which sinus should be laid bare? Given a double otitis, the side showing the more serious symptoms is the side that should be first opened.

The conclusion seems evident that we must be prepared to see an occasional suppurative otitis media infect distant structures. When this occurs without other discoverable foci, we must conclude that the infection has traveled by the usual

route, and we are justified in exenterating the mastoid cells and laying bare the lateral sinus wall, without waiting for any of the usual intervening symptoms of mastoiditis and lateral sinus thrombosis.

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LXIV.

CAVERNOUS SINUS THROMBOSIS.

By Claude T. Uren, M. D.,

Omaha.

The cavernous sinus is an irregular network on the right and left side of the sella turcica and is covered by a dural fold. It surrounds the internal carotid artery and the abducens nerve. The oculomotor, the trochlear, the maxillary and the ophthalmic division of the trifacial nerve are imbedded in the walls of the sinus and, according to Schaeffer, when the sinus is elongated dorsally the mandibular division of the trifacial nerve is in a like position. The Gasserian ganglion is in intimate relationship to the walls of the sinus. Both cavernous sinuses are connected with the circular sinus. The superior ophthalmic vein, receiving the ethmoid veins and connected through the angular vein with the veins of the face, ends in the cavernous sinus. A branch of the angular vein to the pterygoid venus plexus anastomoses with the facial vein and with the pharyngeal veins, and is connected with the cavernous sinus through the foramen ovale.

With this brief anatomic introduction the writer wishes to report two typical cases of cavernous sinus thrombosis which he saw in consultation.

Case 1.—Mrs. D. B., aged 50, was admitted to the hospital August 24, 1924, with the following history:

On August 18th, eleven days before admittance, the patient developed a severe cold in the head with headache. The pain was very severe over the right cheek and right eye. The patient vomited; thinking she was bilious, she took a cathartic, which relieved her vomiting.

Five days later, because of persistent pain in neck and face, the patient consulted her family doctor, who advised removal of two abscessed teeth. A lower right molar and upper left molar were removed the following day. That night her neck became stiff and she had fever.

Two days after removal of the teeth she developed diplopia; there was paralysis of the right external rectus muscle. Pain in the neck became more severe. Temperature 102. White blood cell count 15,000.

The following day a rash appeared over her entire body; white cell count 16,000; pain persisted.

The next day, which was the tenth day of the disease and the day before coming to the hospital, the right eye was completely fixed and bulging and the tension was increased. White blood cell count 18,000; 90 per cent polymorphonuclears. Spinal fluid, 100 cells. No signs of true meningitis. Retina clear.

On the eleventh day of the disease the patient was admitted to the hospital where a consultation was held.

The ophthalmologist's report: Right eyeball protruding; edema of lid and conjunctiva. Eyeball fixed; unable to move eye in any direction. Pupillary reaction nil; tension slightly increased. Vision good. Cornea, aqueous chambers, iris, lens and eye grounds negative. L. B. B.

Surgeon's report: Right eye hard and fixed. Pupil dilated. Edema right eye and right side of face. Evidently phlegmon in the orbit. Recommend opening of the tissues around the eye and sinuses. G. F. S.

Rhinologist's report: Herpes around ala and vestibule, right nares. No pus in the nose. Sinuses clear on transillumination. No polyps or hypertrophies of nasal mucosa. Throat negative. Ears negative. Diagnosis: Cavernous sinus thrombosis.

Radiologist's report: All sinuses clear. No demonstrable pathology in mastoid areas. J. F. K.

Laboratory: White cell count 22,450; 88 per cent polymorphonuclears. Urine 1025, acid, albumin one plus, sugar negative. Temperature 101.8°.

The following day the swelling of right eye and right side of face and back of right ear increased. Temperature climbed to 103.2°. No signs of true meningitis. Reexamination of sinuses by X-ray. Right sphenoid sinus opaque. A. F. T.

The following day, the thirteenth day of the disease, the patient was apparently in coma. Temperature, 103.6°.

Upon request of the family and the internist in charge of the case, the following operation was done under local anes-

thesia; cocain mud applied to sphenopalatine ganglion. Exploratory opening of right sphenoid and posterior ethmoid sinuses.

The right middle turbinate was removed because of a markedly deviated septum, which obstructed any possible view of the sphenoid area. The sphenoid osteum was enlarged down to the floor of the sinus. The posterior ethmoid cells were opened and polypoid lining removed. An exploratory opening was made into the orbital cavity through the posterior ethmoid area.

Findings: Walls of sphenoid sinus edematous; no pus. Very large posterior ethmoid cell lying above and external to sphenoid sinus; lining membrane of cells polypoid. No pus in ethmoids. No escape of pus from orbital cavity. The patient was returned to her room and expired in two hours.

Autopsy—B. C. R.: Body of a white female. There is edema and redness about the right eye and over the prominence of the right malar bone. There is an old scar over each anterior chest and both breasts have been amputated. There are three old laparotomy incisions in the right upper quadrant of the abdomen. Autopsy limited to the head.

There are no changes of the deep scalp tissues, calvarium and on the outside of the dura. There is a fibrinous exudate over the convex surface of the anterior half of the cerebrum extending along the Sylvian fossa to the base of the brain. There are pus, blood and necrotic tissue in the sella turcica with partial destruction of the pituitary gland. The dura over the right side of the sella turcica is dark red, and on opening into the right cavernous sinus there is found a large septic thrombus occluding this vessel throughout its course and extending across the midline through the circular sinus, whose walls are necrotic. The left cavernous sinus is partially blocked with a similar septic thrombus. There is a necrosis of the floor of the sella turcica, with a hole leading into the left sphenoid sinus which contains mucus. The right sphenoid sinus contains blood. A probe introduced through the nose leads directly into it. The posterior ethmoids on the right are very large and contain considerable blood. On opening through the posterior plate of the orbit there is a gush of blood and pus

at the junction of the ophthalmic veins with the right sinus cavernous, but no pus within the orbit proper. There is a hole in the inner wall of the orbit about 0.5 cm. in diameter. There are no changes of the frontal sinuses and the maxillary antrum on the left and the remaining venous sinuses of the dura. There is bloody material in the right maxillary sinus. There are no changes of the middle internal ears and remaining dural sinuses.

Anatomic diagnosis: Acute septic thrombosis of the right sinus cavernosus, with extension through the overlying dura; acute fibrinous leptomeningitis at the right base and up over the convex surface of the right cerebrum; septic thrombosis of the circular sinus; acute exudative hypophysitis; septic thrombosis of the left cavernous sinus; edema of the soft tissues above the right eye due to obstruction of the right cavernous sinus; old surgical removal of both breasts; three old laparotomy wounds.

Case 2.—Mrs. C. A., aged 18, was admitted to the hospital August 16, 1924, because of painful swelling of face and eyes.

Five days previous to admittance to hospital the patient developed a swelling over the left nasal bone which was diagnosed a boil by her physician, who lanced the swelling. There was little discharge of pus and the "core" remained.

The following day she complained of pain in the right axilla. Her right chest was strapped. The second day the patient became weak, fainted and became unconscious.

The following day she had terrific pains in her head and face and a swelling of the left eye.

Examination by otolaryngologist, August 18, 1924: Marked exophthalmos left eye. Slight exophthalmos right eye. Ptosis and marked edema left eye. Left eye fixed and immovable. Left side of face and neck swollen and hot. Nose negative except for small ulcerated area over left nasal bone. Sinuses negative. Small amount of pus in nasopharynx. Vincent's infection of gums. Herpes on lips and around vestibule of nose. Diagnosis: Cavernous sinus thrombosis.

Examination by internist, August 18, 1924: Marked edema and swelling right eye. Diagnosis: Cavernous sinus thrombosis.

A. S.

Examination by ophthalmologist, August 24, 1924: Left eyeball protruding. Edema lids and ocular conjunctiva. Eyeball fixed. Pupil small and inactive. Fundus examination could not be made. L. L. B.

Laboratory, August 19, 1924: Leucocytes 10,200, 90 per cent polymorphonuclears. Urine neutral, 1026. Albumin one plus.

The patient suffered a great deal of pain in the eye and head. She vomited frequently.

August 18, 1924. Twenty-five cc. mercurochrome, 220 soluble, 1 per cent solution, was given intravenously. This was repeated every second or third day, six doses in all. There was very little change in the patient's condition—pain, vomiting, a fever of 101 to 102 degrees continued. A slight chill and marked diarrhea followed each intravenous dose of mercurochrome.

August 21, 1924. The swelling in left eye subsided somewhat. At the same time the right eye became swollen and edematous. This swelling never reached the proportions of the swelling in the left eye. On this date the patient became delirious. This delirium continued with short lucid intervals until death, which occurred August 29, 1924, two weeks after the patient entered the hospital.

In the case of Mrs. D. B., the origin of the infected sinus must be considered in the tooth, and in the case of Mrs. C. A. the origin was a carbuncle on the side of the nose.

In a review of thirty cases of cavernous sinus thrombosis reported in the literature, the origin of the infection was: Suppurative middle ear with involvement of lateral sinus and extension to cavernous—thirteen cases. Suppurative middle ear, thrombosis of superior petrosal sinus and extension to cavernous sinus without involvement of lateral sinus—one case. Suppuration within the nose, sinuses not mentioned—five cases. Suppurative sphenoid sinuses—two cases. Suppurative frontal sinus—one case. Carbuncle on the nose—two cases. Suppurative tooth infection—one case. Tonsillitis—one case. Tuberculosis, general—one case. Lymphocytoma originating in mesentery—one case. Marasma—one case. Primary in cavernous sinus—one case.

In the case of thrombophlebitis of the cavernous sinuses following an infection of dental origin, reported by Lauret, the infection could be traced, step by step, to the inferior dental veins.

The local spmptoms are of two kinds, congestion of the orbit and symptoms caused by involvement of the adjoining nerves. The congestion of the orbit is characterized by edema of the eyelids and conjunctiva as well as by exophthalmos. There may also be congestion of the forehead, the nose, cheeks and neck. Nosebleed occurs in some cases. The second group of local symptoms consists of paresis of the oculomotor and abducens, abnormal position of the eyeballs and ptosis.

Pain is an early symptom. It is partly neuralgic in character and partly headache localized to the forehead and occiput.

Herpes of the nasal ala on same side of lesion was noted in both cases here reported. This symptom is due to an irritation of the Gasserian ganglion.

Pyemic fever, often accompanied by attacks of rigor, is a general symptom.

Diagnosis of thrombophlebitis of cavernous sinus when well advanced does not, as a rule, present much difficulty. However, in the case of Mrs. D. B., consultants did not agree to a diagnosis until exploration of the ethmoid and sphenoid sinuses and the orbit showed them to be free from pus. A similar case, where both ethmoid and frontal sinuses were opened and no pus found and autopsy showed thrombophlebitis of the cavernous sinus, was reported by V. Uchermann.

Another case in which the maxillary, ethmoid and sphenoid sinuses were opened and no pus found, but mention made of ulceration of posterior wall of sphenoid sinus, is reported by Hans Key-Aberg. Autopsy revealed a bilateral thrombosis of the cavernous sinus.

Carl Behr tries to differentiate the symptomatology of an orbital disease of the motor nerves, on the one hand, from that of a lesion in the superior orbital fissure and the cavernous sinus on the other. In the latter two sites of the disease there is a myosis, which Behr observed as a manifestation of a simultaneous paralysis of the sympathetic. The latter occurs in the orbital disease only if there is an extraordinarily extensive process. As the oculomotor nerve represents a central

neuron in the fissure (because of switching into the ciliary ganglion) and the sympathetic a peripheral neuron, the tonus of the latter is completely lacking, and therefore myosis occurs instead of moderate mydriasis.

Therefore exophthalmos with paralysis of all the eye muscles, and with myosis present in spite of absolute pupil rigidity, indicates a lesion in the region of the cavernous sinus or the superior orbital fissure.

The prognosis in cavernous sinus thrombosis is very unfavorable. Most cases terminate in death. However, a sufficient number of recoveries have been recorded without direct attack to encourage us to use every available measure to that end. Spontaneous recovery occurred in fourteen of Dwight and Germain's 182 collected cases.

The literature has very little to offer in the treatment of cavernous sinus thrombosis. Mercurochrome, 220 soluble, 1 per cent, was given intravenously in one case here reported. As noted above, the patient had a severe reaction (chills and diarrhea) following each injection. After the second injection the swelling of the left eye subsided somewhat, but there was no other appreciable change in the patient's condition.

In conclusion, the author reports these two cases of cavernous sinus thrombosis to add to the rather small number reported in the literature. He calls attention to herpes of the nose on the affected side, which he has not seen mentioned in the symptomatology of cavernous sinus thrombosis.

LXV.

CORYZA AND CHLORINE GAS.

By Alan D. Knisely, M. D.,

Lima, Ohio.

That interest in the cause and relief of the common cold has assumed momentous proportions the past several years is evidenced by numerous articles in the lay press and in magazines,[1] by a questionnaire issued from the Surgeon General's office[2] and by a symposium read before the thirty-first annual meeting of the Laryngological, Rhinological and Otological Association.[3]

When an economic consideration is given to the acute upper respiratory tract infections, the deductions in financial figures, physical discomfiture and disability, are appalling. Sickness is one of the most serious taxes levied against industry today. Time lost on account of sickness in 1923 was equivalent to the full working time of a million workers. One large manufacturing concern estimates its losses per annum, per person employed, at $125.00, of which $60.00 is borne by the company and $65.00 by the employee and his family in lost wages, medical expenses, etc.[4]

A cigarmaking company employing 2,500 men and women proved, by giving first aid to minor ills as well as minor injuries, that their loss in days of employment was reduced 40 per cent in comparison with previous months.

A locomotive building company found there were more days lost by employees due to illness than to accidents.

The Public Health Service, after a recent survey, announced that 90 per cent of the population of the United States has a cold once a year, and that 80 per cent of all illnesses is due to common respiratory infections. A study and careful check up, covering 28 months, in the same territory, including 7,200 people, proved each person to be sick 1.13 times a year, and of such illnesses 67 were due to infections of the upper respiratory tract, acute in character, and 46 to all other causes combined.[5]

A survey, in the public schools of a midwestern city of 55,000 inhabitants, to determine the percentage of absenteeism due to common colds, resulted as follows: January, 48.3 per cent; February, 45.2 per cent; March, 43.25 per cent—an average of 45.58 per cent of the absenteeism of the first quarter of the scholastic year due to colds.[7]

(Comment.—Uninterrupted school attendance is necessary for successful educational purposes. There is a tremendous loss in industrial production due to the loss of days' labor, a stupendous financial loss to capital and labor.)

The cause of common colds is without doubt due to the implanting of bacteria in the mucosa of the upper respiratory tract, the propagation of these bacteria and their production of toxic byproducts in sufficient amount as to break down the local resistance of the region invaded and the liberation into the general circulation of toxemias in sufficient amount to disturb the normal physiology.

Moist droplets, impregnated with pathogenic bacteria, sprayed by coughing, sneezing or vocalization from an infected respiratory tract coughed up by the recipient, is the most feasible method of disseminating common colds.

The bacteriology of the common cold varies in different localities and at different seasons; sporadic infections showing entirely different cultures may exist in communities not far remote. Again, the same infection may be endemic.

The disseminated infective material may be in a pure culture or mixed, or may become mixed after implantation, but a predominating bacteria always exists.

Among the common cold producers we find the pneumococcus, streptococcus, staphylococcus, bacillus influenzæ, micrococcus catarrhalis, Friedlander's bacillus and a small anaerobic organism that experimentally produces coryza symptoms in lower animals which has been discovered by Miss Tunicliff.[8]

The chilling of certain areas, such as back of neck or the ankles, resulting in a moderate hyperemia of the chilled part and a temporary mild ischemia of the mucosa surfaces of the upper respiratory tract, thereby lowering the resistance of the nasal mucosa momentarily, offer an excellent opportunity for the already deposited bacteria to become implanted in the epithelial interspaces and the beginning of the common cold. A

deviation in the normal physiologic process, however slight, due to an overload in the intestinal tract of proteins or carbohydrates, any slight change in the blood chemistry, tend to lower the resistance of the nasal or bronchial mucous surfaces and aid the invading bacteria in gaining a foothold. The virulency of the invading bacteria and the degree of resistance offered by the invaded individual govern in direct proportion the severity of the cold.

The treatment of these acute respiratory tract infections has included almost all recognized or known drugs and every conceivable manner of therapy, with varying degrees of results; the most efficient one of these numerous remedies, that of complete rest, receiving the least consideration.

A series of 547 cases of common colds of different location in the upper respiratory tract, with a high percentage of cures, was reported by Drs. Veeder and Sawyer.[9] They offered a plan of treatment which was not entirely new, but which was refined and placed in a usable form and offered a reasonably rational treatment for a malady long in need of serious study.

A series of 900 cases with a large percentage of successful results is reported by Dr. Gilchrist,[10] who mentions that the use of chlorine gas dilute, in the treatment of respiratory tract infections, was experimented with by a Dr. Wallace over a century ago, but at that time such concentration of the gas in the inspired air as would act as a bactericide and at the same time not do damage to the mucosa was not found.

A French physician found chronic bronchial infections were materially benefited and phthisic patients were relieved of some of their disagreeable symptoms, when living near a bleaching plant using chlorine gas as a bleaching agent, near St. Denis, France, in 1834.[11]

Jones and Garoffalo reported 220 patients treated with chlorine gas with eight complete cures, 115 improved cases.[12] The bacterial studies made by Marjorie B. Patterson and her deductions tend to prove that a concentration of chlorine gas of 0.015 mg. per liter of air is not sufficient to render sterile an infected upper respiratory tract.[13]

A report by Diehl states that 23.6 per cent of the patients with rhinitis treated with chlorine recovered within one day after the treatment, compared with 6.7 per cent of the control

series, and 19.5 per cent of the entire group recovered within one day after treatment, compared with 12.5 per cent of the control series. The percentage of recoveries within three days after treatment is almost as large in the control series as in the chlorine series, 47.9 per cent, as compared with 51.5 per cent, and he states that benefit is experienced within the first 24 hours, if any is to be experienced.[14]

A chlorine chamber was installed in the Capitol building in Washington and chlorine gas treatments given to a number of prominent government officers. No statistics are available on these cases, but the Representative from my district took treatments on three successive days without any relief.

Immediately following the publication of Drs. Veeder and Sawyer's article detailing the treatment of colds with chlorine gas, in the J. A. M. A. of March 8, 1924, I made an effort to obtain one of the chlorine producing machines, as described, but was informed by Wallace and Tiernay of New York City that they were not producing the machines for distribution at that time. I interested a local commercial chemist, and he outfitted a chamber containing 1,200 cubic feet of space, supplying the chlorine gas in his laboratory adjoining and delivering it into the treatment chamber in concentration varying between 0.012 and 0.02 mg. per liter of air, an electric fan keeping the gas and air in circulation.

I selected as candidates for the gas treatments patients from my private practice whom I had on previous occasions treated for colds, using them as autocontrols. All cases were examined as to condition of the mucosa of the upper respiratory tract, and if turbinates were swollen and congested they were deturgessed with a spray of 1:8,000 adrenalin chlorid, in order to give free space to the chlorine gas in air.

Two smears were taken from the epipharynx, via the nares, of each case and swabbed on slanted bouillon in culture tubes. One tube was immediately sealed and the other tube given the patient as he entered the gas chamber. At the end of the hour in the gas chamber the second tube was sealed and both tubes tagged and properly numbered and placed in an incubator for 48 hours, at which time colonies were counted and compared in order to ascertain if the gas inhibited the development of

the smear in the tube (subjected to the gas in the same strength as the patient was exposed to). A careful study and check up was made on these cultures and no variation noted. The number of colonies was no less in the tube subjected to the gas than in the one that was not gassed.

Forty-two cases of rhinitis, with nasopharyngeal symptoms, that I had previously treated with other methods, were given hourly treatments on two successive days. Thirty-three of these reported decided improvement, even to relief of all symptoms, on the second day after treatment. The remaining nine did not obtain such decided improvement. Of the 33 who reported decided improvement, 26 reported within eight days and asked to have some other line of treatment used, claiming that they were relieved for several days and then had a return of the symptoms.

On comparing these 42 patients' previous coryza histories, I found, and they agreed, that the old line of shrinkage with 1:8,000 adrenalin chlorid, followed with 15 minutes of packing with cotton swabs saturated with 25 per cent fresh argyrol solution, gave more dependable results.

In this chamber were treated eight cases of pertussis in varying stages; four cases were improved, three cases showed no change and one was for prophylactic purposes only.

The four improved cases were in the same family and were in the fifth week of whooping with emesis, and all received hourly treatment on four succeeding days and all symptoms ceased. These four cases had, the week prior to gas treatments, been exposed to X-ray treatments of the chest.

Five cases, diagnosed as mild influenza, were treated with good results, patients reporting improvement and no return of the symptoms as late as ten days after last treatment.

Five cases of acute bronchitis gave no signs of improvement after six daily treatments, and four chronic bronchitis cases received no benefit.

A university in a near by city installed a gas chamber in the fall of 1924 and treated the faculty and student body during the winter and spring at a nominal price of 50 cents a treatment.

These cases were not selected or examined as to condition of nasal mucosa, and no bacterial check was made and no controls.

I caused to be prepared a questionnaire on a postcard, with reverse side addressed to me, worded as follows:

"PLEASE CHECK THE SPACES INDICATED.

Were you treated for a cold in your
nose....., throat....., larynx....., lungs.....?
How long in days have you had the cold.....?
How many treatments did you receive.....?
Were you cured....., improved....., no change....., or worse.....?
Kindly place this card in the mail."

Approximately 100 cases were treated before the cards were prepared, and from these cases verbal or telephone reports were received, there being no tabulations made, but the majority reported some relief of their symptoms.

The questionnaire cards returned were as follows:

Nose colds, of two days' duration, one treatment, 224 cases, 108 cured, 82 improved, 34 no change;

Nose colds, 3 days' duration, one treatment, 24 cured, 38 improved;

Nose and throat colds (nasopharyngitis), two days' duration, one treatment, 26 cured, 14 improved, 4 no change;

Nose, throat, larynx (nasopharyngolaryngitis), three days' duration, two treatments, ten cases report improved;

Throat, larynx (pharyngolaryngitis), duration, three days, two treatments, eight cases improved.

A personal interview with the general medical men of the university town revealed that 50 per cent of the cases reported as cured came to them for treatment later; the patients' comments were that the gas treatments had helped them at first, but that later they developed a fresh cold.

During this period my friend, the commercial chemist, opened his chamber to the general public, and he reported to me that he had ceased to keep track of the cases; he treated all kinds of colds and about half of them reported good results, but as the newspaper notices of the chlorine gas treatments of colds ceased his clientele gradually dropped off and he closed the chamber in June, 1925.

A study of the appearance of the nasal and pharyngeal mucosa, before and after the exposure to the chlorine gas, dem-

onstrates a moderate hyperemia of turbinates and an increase in the mucous secretion; the congested feeling of the nose is increased for several hours after the treatment, and then the nose gradually opens up and breathing becomes easier per nares. In the bronchial cases the mucus is much freer about six hours after treatment.

The patients showed varied reactions to the gas; some tolerate the chlorine impregnated air with no discomfiture, others complaining of intense dryness in pharynx and a smothering sensation, and some were so sensitive to the gas that they were unable to tolerate the full hour of treatment.

I have used the chlorine gas in ampules and obtained results that compare very favorably with the results obtained in a gas chamber, varying with the personal equation of patients' ability to properly select the right sized room, to have it dry, keep the air in circulation, liberate the gas saturated liquid media in proper amounts and at the right time. If these are properly performed results will be as good as any other method.

Summary: Recognizing the need of some remedy that will abate or shorten the attack and lessen the severity of the acute symptoms of common colds, the chlorine gas suggestion met with a hearty reception, but the results have not borne out or substantiated the glowing offers of the early investigators.

Comment: While reports from other sources have promised much and statistics appeared encouraging, I have failed to find as high a percentage of cures as some of the investigators claim. Granting that chlorine is a powerful bactericide and that in laboratory tests it will render inert certain bacteria when the gas is used in a concentration of 0.015 gm. per liter of air, yet conditions in the passageways of the upper respiratory tract cannot be compared to laboratory conditions and do not give the same results, as the gas does not penetrate into the minute folds and crevices in the mucosa of the lateral walls of the upper pharynx and the recesses of the lower pharynx, nor does it penetrate into the accessory sinuses of the nose, and in all of these places are the invading bacteria.

Dr. Veeder reports, since his original article, that the bactericidal action of the chlorine is not the only effective agent at work to destroy bacteria. As the chlorine acts as a high

oxidizer, hypochlorous acid is formed by the union of the chlorine and the water in the mucus, which in turn is reduced to hydrochloric acid with the liberation of nascent oxygen, in this way decomposing toxins and reducing the toxicity of organisms.[15]

I cannot see that we are any nearer an ideal treatment for relief of the common cold than prior to the advent of chlorine gas, though a stimulus has been applied and workers are striving for a remedy that will relieve the world of a heavy burden.

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LXVI.

BRONCHOSCOPIC STUDIES OF SOME CONDITIONS OF THE RESPIRATORY TRACT.

By Simon Jesberg, M. D.,

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Until comparatively recently the chief activity in bronchoscopy has been concerned with the removal of foreign bodies from the air passages. This is probably because the novice in the field of peroral endoscopy has his first adventures with this type of cases, but as experience widens his field he soon finds many new angles and phases suggested by the conditions he encounters.

Today the trend in bronchoscopic literature indicates that interest is being aroused in a wide range of respiratory lesions other than foreign bodies, in which endoscopy plays an important part.

In an analysis of 341 endoscopic examinations and treatments, at the Bronchoscopic Clinic of the Eye and Ear Hospital of Los Angeles in a period of twelve months, only thirteen were concerned with the removal of foreign bodies.

It is the purpose of this paper to present some pathologic conditions encountered, with special reference to, variations in, and causes of, obstruction of the air passages.

A consideration of certain basic principles of lung X-ray findings is essential to the bronchoscopist if he is to arrive at a conception of the mechanics involved and the conditions governing changes in the air passages. Decreased radiability of lung tissue is due to the presence of infiltrations, secretions or lung compression. Increased radiability is due to emphysema or cavitation.

When a bronchus is completely obstructed, the confined air is absorbed, with the result that the lung collapses—i. e., atelectasis occurs. If the obstruction is only partial, changes occur which vary with the degree of obstruction and the length of time in which this obstruction continues.

The air passages dilate during inspiration and contract during expiration, hence a partial obstruction of the lumen is more complete during the expiratory effort. Upon this principle depends the phenomenon of "air trapping," which is of paramount value in the localization of nonopaque foreign bodies and may also be present in bronchial obstruction from other causes.

In a typical case of "air trapping," due to the obstruction of a main bronchus, for example, the right, a roentgenogram taken at the height of inspiration shows both lungs overdistended, and probably equal in radiability; both diaphragms are depressed and the mediastinal shadow is in the normal position. A plate taken at the end of expiration shows the left lung deflated and the left diaphragm elevated, the right lung unchanged, being still overdistended, the right diaphragm depressed and the mediastinal shadow displaced to the left.

Complete obstruction during expiration is not always present, all degrees of "air trapping" may occur, varying according to the size of the obstructing body in relation to the size of the lumen in which it rests. However, the air space around the obstruction is always greater during inspiration than during expiration.

In minor degrees of bronchial obstruction, the trapping of air is less evident and consequently more difficult to recognize. This less retarded emptying of the distended lung is better observed by means of the fluoroscope than by the roentgenogram.

In a case of partial obstruction of the left main bronchus by a fragment of a nut kernel, eleven roentgenograms were taken before air trapping was definitely demonstrated. Dr. R. G. Karshner quickly detected the condition under the fluoroscope, but he noted that air trapping was present only at times. This was made clear to us when the foreign body was examined after its removal. It proved to be a small fragment of nut kernel, which was partially encased in the husk or skin, a part of which was loosely attached, and, moving back and forth in the respiratory blast, presented varying degrees of obstruction, at times very little and at times almost complete. A loud expiratory wheeze could be heard with the stethoscope during nearly every expiration.

A nine-year-old son of a physician choked while eating peanut candy, eight days before coming to the clinic. Aside from a slight cough he had no other symptoms. Several roentgenograms had been taken, none of which demonstrated any abnormality. The father, during frequent chest examinations, had noted that now and then there was an expiratory wheeze over the right hilum, and again breath sounds were diminished in the right middle lobe. He also noted that he could cause a change in these auscultatory findings by thumping the patient on the back and by change of position. Dr. Karshner, after watching the patient's respirations for some time with the fluoroscope, was able to detect slight evidence of air trapping on the right. This taken with auscultatory findings led to a diagnosis, and the fragments of peanut were removed from the right bronchus with uneventful recovery.

When partial obstruction of the trachea exists, "air trapping" is present in both lungs. The mediastinal viscera are not laterally displaced; the diaphragm is depressed on both sides and the lungs are overdistended, both during expiration and inspiration. Any considerable degree of tracheal obstruction necessarily causes severe dyspnea, and urgent attention is demanded to prevent suffocation. With such severe dyspnea, time for X-ray studies is usually not permitted. A two-year-old child with a peanut in the trachea nearly died of suffocation while time was being taken for X-ray study. Bronchoscopic removal of the peanut was accomplished barely in time to save the child's life.

Any patient presenting dyspnea or air trapping for which adequate explanation cannot be elicited by the attending physician should be suspected of harboring a foreign body in the air passages. Naturally such cases eventually fall into the hands of the bronchoscopist, who is confronted with the responsibility of diagnosis and treatment. Oftentimes in our experience the cause of the patient's difficulty has proven to be other than foreign body obstruction, and it is a group of such cases that I have chosen to present.

1. Obstruction of the Left Main Bronchus by a Polyp.—A six-year-old boy, referred by Dr. Hugh Berkley with diagnosis of obstruction of the right bronchus. Dr. Berkley suspected a foreign body. In March, 1925, this patient developed

pertussis, from the symptoms of which he mainly recovered, continued cough and expiratory wheeze persisting. X-ray showed partial air trapping of the left lung. September 12, 1925, bronchoscopic examination with butyn anesthesia: in the left bronchus a soft, round, pedunculated tumor was found, which came into view during expiration and disappeared into the upper lobe bronchus during inspiration. It was difficult to grasp the growth, due to its excursions. Finally it was removed piecemeal with a straight grasping forceps. Twelve days later a second bronchoscopic examination showed only a small granular base at the orifice of the left upper lobe bronchus. Fifty per cent silver nitrate was applied. There has been no further respiratory disturbance noted in this patient. Biopsy report by Drs. Brem, Zieler and Hammack diagnosed a polyp.

2. Obstruction of the Right Bronchus by Peribronchial Compression.—A girl, eighteen months old, with clinical diagnosis of bronchopneumonia. X-ray showed air trapping and some infiltration of the right lung. Bronchoscopic examination, September 8th, showed a widening of the carina and closure of the lumen of the right bronchus. The mucosa appeared normal and there was no abnormal secretion. An attempt was made to dilate the closed bronchus, which failed. A closed forceps could be passed one cm. further. Impression: Bronchus closed due to pressure from some mediastinal mass. On September 10th a second bronchoscopy was made with the same findings. On September 11th, an X-ray of the chest showed no air trapping. However, two days later air trapping was again present. The patient reentered the hospital April 1, 1926, when the final diagnosis of tuberculous peritonitis was made. At this time X-ray of the chest showed pulmonary tuberculosis. The patient is still alive. A diagnosis of closure of the right bronchus by pressure, due to tuberculous mediastinal glands, is probably correct.

3. Obstruction of the Left Bronchus, Mediastinal Compression.—A nine-months-old infant, well developed and nourished, had had more or less dyspnea since birth. X-ray showed air trapping in the left bronchus and a large thymic shadow. Bronchoscopy with butyn anesthesia: the mucosa of the larynx and trachea is congested; anterior bulge of the tracheal wall

indicates thymic enlargement. The lumen of the left bronchus is closed one cm. from the carina. Forceps passed through the swollen lumen discovered no foreign body. Within twenty-four hours a tracheotomy became necessary on account of dyspnea. Next day the X-ray still showed air trapping of the left lung. Lower bronchoscopic examination five days after the first showed no change, except that the tube could be passed two cm. into the left bronchus. Air entered and passed from the lung while the tube was in the bronchus. No foreign body was discovered. The impression of this case is that there is a closure of the left bronchus due to mediastinal compression. Possibly the large thymus is in some way responsible for the condition.

4. Obstruction of the Right Main Bronchus by Swollen Mucosa and Pressure, a Foreign Body Being in the Left Main Bronchus.—A boy, age four and a half years, was brought to the hospital January 15, 1926, with severe dyspnea. History of aspirating a nut kernel one week before. X-ray showed air trapping both lungs. Bronchoscopic examination showed the left bronchus obstructed by a fragment of nut kernel. The lumen of the right bronchus was almost closed by swollen mucosa. Exploration of the right bronchus showed no foreign body. Dyspnea was relieved by removal of the foreign body. January 18th, the second bronchoscopic examination showed the trachea and bronchi normal. No further respiratory difficulty has been encountered. Impressions: The left bronchus was obstructed by a foreign body; obstruction of the right bronchus was due to inflammatory reaction; venous engorgement and lateral displacement were caused by air trapping of the left lung. This condition corrected itself after the removal of the foreign body.

5. Obstruction of the Left Bronchus Due to Tuberculous Granuloma (Lupus).—A woman 20 months ago developed an acute respiratory condition, which was considered to be whooping cough. After eight weeks symptoms subsided, except cough with mucopurulent sputum persisted. This cough has persisted to date; the health otherwise is good; she has not lost in weight. Tubercle bacilli have been found in the sputum. Dr. Roland S. Cummings detected signs of obstruction in the left bronchus. Bronchoscopic examination showed a flat soft gran-

ular growth occupying the left lower wall of the trachea for a distance of 4 cm., extending down to and filling the left bronchus. A section was taken and examined histologically; it showed tuberculous granuloma with giant cells.

6. Obstruction of the Trachea and Bronchi Due to Viscid Secretion During Chickenpox.—A four-year-old girl with chronic laryngeal stenosis and wearing a tracheotomy tube developed chickenpox. The second day after appearance of skin eruption dyspnea developed, which was not relieved by changes of the tracheal tube; mucus and secretions were profuse. Within twenty-four hours after beginning of these respiratory symptoms breathing became almost impossible; cyanosis became marked and the patient comatose. A bronchoscope was passed through the tracheal wound, and the walls of the trachea and bronchi were found to be covered with a thick gummy secretion; this was removed by forceps and a suction canula, resulting in free breathing. This cleaning was again done twelve hours later, after which the child made a complete recovery from her dyspnea.

7. Obstruction of the Right Bronchus and Collapse of the Right Lung During Bronchopneumonia.—On March 29, 1925, an infant of ten months entered the hospital with bronchopneumonia. X-ray showed complete collapse of the right lung. Bronchoscopy, March 30th, showed closure of the right bronchus. Gentle dilatation and aspiration of mucus opened the lumen of the bronchus. Air entered the lung, and X-ray next day showed the lung normally filled with air.

8. Partial Obstruction of the Left Bronchus Due to Traction and Pressure by Pericarditis.—A four-year-old girl in the Children's Hospital with a complication of conditions, one of which was signs of obstruction of the left bronchus and varying degrees of lung inflammation. Bronchoscopic examination showed a widened carina, the left bronchus was displaced upwards and compressed from below; no intrabronchial lesion was found. At autopsy, some weeks later, pericarditis was demonstrated. The bronchial compression had been due to displacement by the enlarged pericardial sac.

9. Tracheobronchial Obstruction Due to Diphtheria.—A three-year-old boy was brought to the clinic of the Children's Hospital. He had been sick for eight days with respiratory

symptoms, dyspnea becoming marked the last two days, and weakness marked the last twenty-four hours. There was no history of foreign body. Chest examination indicated obstruction of the right bronchus. Radiogram of the chest showed obstruction of the right bronchus and infiltration of the right lung. Endoscopic examination showed a membrane in the trachea which extended into the right bronchus. Enough of this was removed to partially free the air passage. The patient died shortly from toxemia. Culture from the tracheal membrane was positive for diphtheria.

10. Atalectasis of the Newborn.—This patient, a full term baby, with normal delivery, failed to establish normal breathing; respiration was rapid and shallow. Cyanosis became more marked. Radiogram showed collapse of the right lung. As hurried preparations were being made for bronchoscopy the child quit breathing. A 3 mm. tube was passed and oxygen administered; adrenalin was injected in the heart, but efforts at resuscitation failed. Autopsy was not permitted. Having had several occasions to make bronchoscopic examinations of the newborn, we discovered that the standard 4 mm. tube is too large; the 3 mm. tube above mentioned has too small a lumen to be practical. A tube more suitable for the newborn is now in course of construction. If this case had been brought earlier for bronchoscopy it is possible that a less tragic termination would have resulted.

11. Spontaneous Pneumothorax in a Young Child.—A two-year old girl, late in the evening of a holiday, was brought into the California Hospital. She had had a respiratory difficulty for three days, which had begun suddenly. Dyspnea had become progressively worse, till at the time of examination cyanosis was marked. A hurried examination showed the left chest distended and stationary; marked increase of resonance to percussion and almost absent breath sounds; the heart was markedly displaced to the right. A diagnosis of air trapping of the left lung was made. While waiting for the roentgenologist to arrive at the hospital the child died. Efforts at resuscitation failed. A plate taken immediately after death showed a pneumothorax on the left, with thoracic viscera displaced to the right, compressing the right lung. Investigation into the past history showed that the child had been in good health.

The father stated that the child was in the habit of carrying a wooden toothpick in her mouth much of the time. There was no history of aspiration of a foreign body. A postmortem endoscopic examination was done, but aside from displacement of the trachea and bronchi to the right no abnormality was found. Further postmortem examination was not permitted.

The possibilities of peroral endoscopy are by no means yet exhausted. One engaged in this work is constantly meeting conditions that make him realize that many new experiences are still in store for him, and his interest in this field is kept alert by an endless variety of adventures.

The general profession is becoming more aware of some of the possibilities, and with their cooperation and that of the internist and radiologist peroral endoscopy will be brought into its wider field of usefulness.

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LXVII.

WHAT IS WRONG WITH THE TONSIL OPERATION?*

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More than 2,000 years ago Celsus described his finger enucleation of the tonsils. Since this time the surgical removal of the faucial tonsils has been the subject of most prolific literary effort. Indeed, volumes have been written upon this topic, mostly during the last decade, the preponderance of the work dealing with methods of removal. Is it any wonder that the average physician has thought only of removing the tonsil, instead of carefully inquiring as to whether or not it was really the cause of the disability? And yet the literature has been rich in work of the most scientific nature on the subject. The investigations of Loeb, Halstead and Shambaugh have thrown light on the tonsil as a possible focus of infection. Friedburg has shown the tonsil as the site of trouble in diphtheria carriers, while Selfridge has done valuable work on the relations between the tonsil and the ductless glands. With all this mass of work upon the subject, some extremely valuable and some quite the reverse, it is not my purpose to attempt to offer anything new or original, or to write any comprehensive review. My object is simply to make a few observations upon the practical application of the subject. My inspiration has been a letter received from a pediatrician, in which he said that he had become quite disappointed in the tonsil operation and a bit skeptical of its results. It is my purpose to try to answer and to allay the doubts expressed in this communication. That this is a subject of quite widespread interest is very evident when one glances at the literature of the past few years. Kaiser's study of the effect of tonsillectomy on the general health of 1,200 children, as compared with an equal number

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not operated upon, has given us all food for thought. His findings were, to an alarmingly large extent, disappointing, in so far as effect on general conditions went. Why this dissatisfaction? Are we expecting too much? Are we applying this procedure too broadly as a sort of "cure all," without due regard to individual study? Or is the fault due to the operation?

Just as the tonsil operation has been so prolifically written about, so it has been practiced. In this fact, I believe, rests the answer to the above expressed problem. Rather than condemn the procedure, we should criticise its application. Just as the removal of the appendix has become a greatly abused surgical procedure, in many cases of abdominal pain, often regardless of the diagnosis, because it is an operation that is within the ability of the socalled surgeon to do—one that is in his repertoire—even to a greater extent has the removal of the tonsils been the means of ill directed surgical effort. Because it is generally considered an easy operation to do, too often is it resorted to without a careful study of the case in question, to determine if this is the proper procedure. Often, indeed, has the almost wholesale removal of the tonsils become the "slaughter of the innocents." Like the traditional Irishman with the club, it sometimes seems to be the endeavor of the physician to strike down every tonsil encountered, whether pathologic and the indicated cause of the disease or not.

This is sufficiently open to criticism when due to the indifferent or careless attitude of the physician specially trained in throat work and supposedly capable of careful diagnosis in this field. It becomes much more culpable when the general physician, lacking in laryngologic training, starts the process of tonsil elimination wholesale. Once I heard a well known surgeon say that "nowadays anyone is a surgeon who can buy a knife and get someone to lie still for him." I would paraphrase this remark to the effect that "anyone is a throat specialist who owns a tongue depressor and some sort of tonsil instrument, and can get someone to open his mouth." Too many physicians have been concerned only with the question "how," instead of "why." Quite probably this attitude has been due to the mode of postgraduate teaching in vogue up to

the last few years. Students going to the large medical centers put in perhaps an average of six weeks' work, intent only on learning how to do a few operative procedures. They learned how to remove tonsils, possibly; but the more important question of when and why was never answered. There was no time or inclination to study the anatomy, physiology, or the pathology. Operative technic was the sole objective. But now there has been a reformation. Repeated warnings have been sounded for years, and at last otolaryngology has begun to put its teaching on a firm basis. The establishment of the National Board of Otolaryngology is a most important step towards putting this specialty on a higher plane. As a result we will have more real clinicians and fewer operators.

Too often is the tonsil operation considered as a shot in the dark, as a gamble. Too often is the patient advised to have this done with the vague hope, in the physician's mind, that it may be of some help. Sometimes I'll admit that it is necessary to proceed on just these very grounds in the process of elimination, but, in by far the majority of cases, I consider that one should have pretty definite grounds for making a diagnosis of infected tonsils and fairly well defined reasons for expecting benefit from any operative procedure. Neither should we think of this operation as a "cure all," or as a step to be taken when all else fails. In its proper application it is simply an operation for the removal of infected or diseased tissue, and should be performed only when definitely indicated as a probable, or at least possible, cause of the patient's disability. Unless so applied there will be little satisfaction either to the patient or to the physician.

It may be fairly assumed that, to a large degree, the dissatisfaction with the results of the tonsil operation are due to its improper application: to

1. Faulty operative technic;
2. Incorrect diagnosis;

(a) Inattention to predisposing factors;

(b) Lack of careful study and intelligent interpretation of the clinical picture, including history and examination, both general and local;

(c) Ill advised time of operating.

If the tonsil is the cause of the patient's disability and if it can be removed completely without detriment to the patient the result should be satisfactory.

Anatomically the faucial tonsils are placed in the fossæ between the anterior and posterior palatine pillars. They are masses of lymphoid tissue, oval in shape, and vary in size according to their condition and the age of the patient. The anterior pillar, or boundary, is made up of the palatoglossus muscle; the posterior pillar, of the palatopharyngeus muscle. Between these, forming the bed of the fossa, we find the fibers of the superior constrictor and internal pterygoid muscles, while at its base the tonsil is somewhat attached to the fibrous tissue of the tongue. Much has been said about the socalled capsule of the tonsil, but this seems to exist only in some of the literature. Israel is convinced, from his microscopic investigation of 172 tonsils, that any capsule, as ordinarily understood, does not exist. Rather do we have an aponeurotic fascia separating the muscle from the lymphoid tissue with its supporting connective tissue. We may think of the tonsil as a mass of lymphoid tissue, plastered or thrown into this bed between these muscles, a good deal the same as we might throw a snowball at a crevice in a wall and have it stick there. In the same way the adenoid, sometimes called Luschka's tonsil, is a mass of lymphoid tissue in the vault of the nasopharynx, the greater portion being central but spreading out laterally and often filling up the fossæ of Rosenmüller. These, with the lingual tonsil on the base of the tongue, make up Waldeyer's ring of lymphoid tissue. Israel also points out that comparatively few tonsils are removed without some muscle tissue attached. This is easily explained when we recall the situation of the tonsil in regard to its bed. On the epithelial surface we find from ten to twenty crypts, extending deeply into its substance. These may be very irregular, extensively branched, and as Barnes says, with their caliber much greater below the surface than their faucial openings would indicate, making for the accumulation of débris and infected material and for difficulty in drainage. The chief blood supply to the tonsil is from the tonsillar and ascending palatine branches of the facial artery, while the descending palatine, the dorsalis linguæ and the ascending pharyngeal send branches

into its midst. There is a large plexus of veins lying in the wall of the fossa. Despite the work of many investigators in injecting inert coloring matter in the turbinates and gums and finding it in the tonsils, according to Barnes there is no proof of any afferent lymphatics. The efferent lymphatic system drains into the cervical nodes, with the socalled "sentinel gland" at the angle of the jaw. The lingual tonsil, let us remember, is simply a small replica of the faucial. To be sure, the crypts are not as tortuous, for the space will not permit, but histologically it is similar and, more often than is thought, it is of similar pathologic significance.

Nothing can be said, with any degree of certainty, in regard to the function of the tonsil. Its physiology is undoubtedly the same as that of the lymphatic nodules elsewhere in the body. In common with the other lymph nodules it probably has something to do with the production of lymphocytes for the blood stream. The theory of some internal secretion, formed in the tonsil, is only a theory and has no supporting evidence. Much has been written concerning the theory that the tonsil served as a protection against bacterial invasion, but practically nothing has been proven. The tonsil, undoubtedly, may prevent the absorption of bacteria from the crypts into the lymphatics. It may act just as any other lymph gland in holding up organisms and, in this way, has a protective function; but no more so than any cervical gland. Possibly this conception of the tonsil as a lymph gland would be a good one for the profession to keep in mind. If it were considered, surgically, just as a cervical gland is considered, and removed only when there were definite indications, there would be less dissatisfaction all around. This might limit the temptation towards unrestrained efforts at removal because of its accessibility.

According to Barnes, bacteria appear in the crypts of the tonsil 24 to 72 hours after birth, at about the same time as the bacterial invasion of the intestinal canal. Considering the structure of the tonsil with its deep and tortuous crypts, easily occluded at their orifices by inflammatory reaction, is it any wonder that they are so frequently the site of an infectious process, manifesting itself either locally or systemically?

If we will look on the tonsil operation simply as a proper means of eliminating a focus of infection, or diseased, or hypertrophied tissues, which are the cause of disability to the patient; and carefully endeavor to prove this to be the case, rather than relying upon "snap diagnosis," or considering it in the category of a fair gamble, we may assume that any subsequent dissatisfaction will be due either to incorrect diagnosis, or to faulty operative technic. The operation has received so much publicity and is so widely known, even among the laity, that oftentimes we may be almost stampeded into performing it, when the facts hardly justify it. Physicians, and even patients themselves, having heard of improvement following this operation among their friends, sometimes demand that we operate. It is in the same category as the extraction of teeth. Someone has some vague, ill-defined trouble and is advised on all sides to either have their tonsils or their teeth out. In the face of such rash and hasty proceedings, is it any wonder that there is a great deal of unsatisfactory after results?

The mass of work on the tonsil problem has been upon various methods of removal, surgical or otherwise. One could almost say that there were as many different operative procedures as there were operators. All have their advocates and, practically all, their advantages. What is the best operation for one may be quite the reverse in another's hands. We are all agreed that enucleation is the only proper course to pursue. Then the operation which removes the offending tonsil completely, with the greatest degree of safety to the patient and the least trauma, discomfort, and distortion to the throat afterwards, at the hands of any operator, is the best one for him to employ. First, prove in so far as possible that the tonsil should be removed, and then do it by the safest, surest, and least damaging method. Briefly we may divide the operative methods into those which dissect the tonsil either by knife, scissors, or blunt instrument, and complete the removal with the snare; and those which dislocate the tonsil from its bed and then sever its attachment. Personally I believe that both types have their field of usefulness and that the aspect of the individual case should determine just which method to employ, provided the operator is

competent to use more than one. In children, with large so-called "virgin" tonsils, those not tied down with adhesions, or scars of old peritonsillar abscesses, where the tonsil will easily dislocate, I prefer the LaForce instrument, because of the easier convalescence resulting from its use, in my hands. In all of the buried type, those tied down with adhesions, or with histories of old peritonsillar disease; and those, encountered far too often, which have already had a tonsil operation, either tonsillotomy or unsuccessful tonsillectomy, and in cases done under local anesthesia, the sharp knife and snare dissection is my choice. I believe that it is wise to have several different types of tenacula, or seizing forceps, at hand so that the tonsil may be grasped in such a manner as not to distort it before dissection. All cases should be hospitalized and prepared for operation as carefully as for any other surgical procedure. A careful general physical examination should always precede, and this should include the routine laboratory tests, with some investigation as to the coagulation time of the blood. The work at the Massachusetts Eye and Ear Infirmary, where all children under 10 years of age have been routinely examined by X-ray, has shown the presence of an enlarged thymus gland in 7 per cent of their cases, an observation of alarming significance. While this X-ray examination of every child may seem a difficult routine for the average surgeon to follow, its importance cannot be overestimated. We should always consider the possibility of persistent thymus in the young child and routine X-ray examination is the only really safe course to pursue. I have had a small series examined but so far we have not encountered any positive cases. It may be that the type of case going to the charity clinic in the large city is more apt to have a persistent thymus than the type we find in private practice, or in our locality. Those more versed in regard to the endocrines will be better able than I to answer this question.

While most of us routinely employ the preliminary hypodermic medication of morphin and atropin, some men have expressed doubt as to its advisability. Bigelow felt that the patient was safer without this as it might have some inhibitory influence on the pharyngeal and cough reflexes which are protective to the respiratory tree. Patients, whether for local or

general anesthesia, should not be allowed to walk to the operating room, but should be carried there, and during the administration of the anesthetic the room should be quiet and nothing should be allowed to excite the patient. A nitrous oxide and oxygen induction in all ether cases, except the very young, is of great solace to the patient and reduces the amount of ether required.

Despite all that has been written concerning the possibilities of pulmonary complications, due to the position of the patient, I do not believe that this makes very much difference. Certainly statistics do not show this to be a factor. We have yet to learn whether these complications are inspiratory, or embolic. Authorities differ. Probably they occur in both ways. Whatever the route, the main thing in prevention is a skillful, careful operation performed at the proper time. The condition of the patient, the nature of the operation, control of the bleeding and avoidance of the undue trauma are the biggest factors. Bigelow has pointed out that the sitting position is the position of choice for one to cough:—and the cough has been called by Jackson, "the watch dog of the lungs," and is generally considered a means of clearing the trachea, although Myerson's findings in a series of bronchoscopic observations during tonsillectomy showed that blood and saliva do not impart sufficient stimulus to set the cough reflex in motion. He does not feel that the cough is the normal mechanism of expulsion in tonsillectomy under light anesthesia. Possibly the swallow reflex is of more importance, or that later the patients clear the tracheobronchial tree by coughing. At any rate a bit of coughing makes the operator feel somewhat safer. With the patient sitting forward and a careful anesthesia, a most important factor, so that the pharyngeal, or swallow reflex, is not lost, careful technic and the constant use of suction, I consider that we are as safe as possible. There is some difference as to the advisability of the use of suction. Some men, including Large, feel that it predisposes to bleeding. Used carefully, it seems bound to be a decided aid in the operation. I use a suction tube attached to the under side of the tongue depressor. It is always in place, does not directly touch or suck upon the tissuees of the fossæ, and certainly makes for a clear field. All bleeding points

should be controlled and the field should be dry before the operation is considered complete. Hemostats, ligatures and, if necessary, sutures will accomplish this, if one adds patience and persistency to one's armamentarium. Green advocates holding the severed tonsil firmly in the fossa for two minutes after removal, using the tissue juices to facilitate clotting. Jervey makes use of the Cushing brain clamp when ligation is too difficult.

Most every operator has his own pet routine of postoperative treatment, but I doubt if anything has ever been devised which will discount, to any marked degree, the discomfort following this operation. The amount of suffering will, of course, vary with almost every case. Much depends upon the individual, his resistance, the condition of his throat, the type of operation employed and the amount of manipulation involved in the procedure. The least possible amount of sponging during the operation should be insisted upon. Rest in bed, attentive nursing, opiates, or sedatives if required, and an intelligently selected diet, all make for the well being of the patient. Heat applied to the neck is comforting, while the use of warm saline irrigations, as well as a gargle, or mouth wash, of aspirin solution, 20 grains to a glass of warm water, seems to bring relief. Having the nurse lift up the head, putting traction upon the pharyngeal muscles, enables the patient to absorb nourishment and fluids with added ease. Various local applications both immediately after the removal of the tonsils, and later during the convalescence, are advised by many. Compound tincture of benzoin, tincture of iodin, silver nitrate, scarlet red, argyrol, etc., have been so employed, but it is doubtful if there is much if any benefit accruing from these measures. After five or six days, when the pain is largely "muscle soreness," having the patient chew gum exercises these muscles and seems to limber up the tissues with resulting comfort. But at the best we must count on a period of discomfort varying from six days to two weeks. The redeeming feature is the promise of better days ahead and usually the discomfort is sufficient to make the succeeding period bound to seem one of great improvement.

One common source of error is failure to pay attention to the lingual tonsil. Frequently overlooked, it may be as much

at fault as the larger and more evident faucial tonsil. Especially when using the Sluder or LaForce type of instrument, one is apt to leave a bit at the lower pole which, if neglected, may hypertrophy and migrate upward into the fossa, later giving the appearance of a pretty fair sized tonsil. This lower bit, and any evident lingual tonsil, should be removed. This will save quite a lot of later dissatisfaction. Dr. H. E. Thompson has very kindly examined a few lingual tonsils for me. The number available at this time was too meager to make the findings of any real value, but he has reported cultural growths of streptococci, staphylococci, and a gram negative bacillus, showing that we must consider the lingual as well as the faucial tonsil as a possible focus of infection.

Proper preoperative preparation of the patient, insistence upon hospitalization, a trained operating room crew, careful anesthesia with due regard to the preservation of the pharyngeal reflex, with conscientious after-care, all are conducive to a satisfactory result. A great deal of the postoperative criticism is due to incomplete operation, leaving bits of tonsil or adenoid tissue, distortion of the throat, obliteration of the pillars, and damage to the uvula, or soft palate. Due care will eliminate most of these at the hands of the trained operator. Careful inspection of the fossæ, after removal of the tonsil, and especially of the under side of the anterior pillar, will sometimes reveal a bit remaining, when the operator was certain that the tonsil had been removed in toto. Many so-called recurrences are simply due to the hypertrophy of what is really extracapsular (socalled) tonsillar tissue, lymphoid follicles at the base of the tongue, or a part of the lingual tonsil. Care to remove these, if demonstrable, will often solve this difficulty. Sometimes patients who have had their tonsils removed, will subsequently complain of irritative throats. Examination will reveal hypertrophied lateral pharyngeal bands. Apparently Nature sometimes tries to make up for the loss of lymphoid tissue by causing this enlargement. Why this should be we do not know. There may be a dietary factor, or the endocrines may have something to do with it. If we can get at the fundamental cause of this hypertrophy we will accomplish more than can ever be done by way of local applications. Incidentally, patients should not be told

that after a tonsillectomy they will never have a sore throat, for such a statement is apt to breed disappointment and dissatisfaction. They should be given to understand that for some very good reason it is deemed advisable to remove their tonsils: that after this is done this evident focus of infection will be eliminated and that they will not have "tonsillitis." However, they should realize that they are liable to have a sore throat, for the mucosa of the pharynx may become inflamed, or these lateral bands swollen, if sufficient cause is given.

During the past few years we have heard a great deal concerning the treatment of tonsils by X-ray and by radium. There has been much discussion pro and con, but its advocates have given little scientific or clinical proof that this is practical. The X-ray advocates have laid stress on the reduction in size of the tonsil almost entirely. We all must realize that this is of little importance. It is the infective organism within the tonsil that we wish to eliminate. Witherbee, the leading exponent of this method, has been the target of a great deal of criticism, due to his radical statements without clinical backing. McMillan, at the Massachusetts Eye and Ear Infirmary, found that in none of his cases, after ten to twelve exposures, did the lymphoid tissue disappear. The size of the hypertrophied tonsils decreased to about normal. The observations of Shurly were similar except that the decrease in size was only about 50 per cent. Borden has shown by examining tonsils removed, after having been subjected to the X-ray treatment, with the technic advised by Witherbee, that there is no decrease in the infection of the tonsil; in fact, he is inclined to believe that it may be quite the reverse. And may not any shrinking of the tonsil serve to seal the crypts and prevent drainage, thus increasing the amount of infection? Less has been heard about the use of radium, but several competent observers have reported success from its use. Electrocoagulation has not been practiced to any large extent, and little can be said for it at present. But if a tonsil is infected and a source of danger to the patient, the only sensible course to pursue is to remove it thoroughly, with whatever method promises the greatest degree of safety and surety against recurrence of trouble to the patient. No re-

liance can be placed upon the X-ray treatment. In certain cases of bad operative risks, such as "bleeders," the advanced tubercular, or cardiac cases, or in the senile, radium, or even electrocoagulation may be employed to advantage. But our main reliance must be upon the surgical removal, with irradiation limited to cases with marked contraindications to operation.

The dissatisfaction due to faulty operative technic can be remedied by a more careful and critical attitude on the part of both the physician referring the case for operation and on the part of the patient. Too many men are attempting this operation who are unqualified to do it. While they may "get away with it" in the majority of cases, they are bound, sooner or later, to run up against a condition which invites disaster. If no man attempted any surgical procedure who was not qualified and capable of handling any condition which he might find in that given field, the world would be safer for all concerned and surgery would be upon a higher plane. If consulting physicians and patients, or their families, would insist upon the best available operative skill, much of this trouble would be eliminated.

The conditions justifying the removal of the tonsils may be classed under two headings: local conditions in the pharynx, and general conditions in which we may consider the tonsil as a cause of the trouble, or a focus of infection. Under the first we have tonsillitis (repeated attacks); peritonsillar abscess, Vincent's angina, tuberculosis of the tonsil, neoplasm, otitis media, either suppurative or catarrhal, where the tonsil or adenoid condition has a direct effect on the eustachian tube and middle ear; in diphtheria carriers, and sometimes where the tonsils or adenoids are such as to mechanically obstruct breathing. Under the second heading of general conditions which may be caused by a focus of infection in the tonsil, we have: arthritis, acute or chronic endocarditis, myocarditis, pericarditis, chorea, nephritis, acute or chronic; pyelitis, neuritis, infectious jaundice, osteomyelitis, pulmonary gangrene, cervical adenitis, paranasal sinus disease, predisposition to common colds, and chronic toxemias without localized lesions, as well as certain endocrine disorders. Even appendicitis and

peritonitis have been considered as due to tonsillar infection. The possibility of the tonsil being the focus in cases of joint, heart, kidney and glandular disease is seldom lost sight of in these days. The criticism is rather due to the tendency towards jumping at the tonsil as the offender instead of trying to ascertain by careful investigation and study if it is the cause of the disability. Sometimes it is only by the process of exclusion that this is possible. The condition of the teeth and gums, ears, sinuses, gastro-intestinal and genito-urinary tracts must never be lost sight of as possible foci. And if the tonsil be the primary focus of infection we may have recurrences after removal, the infective agent remaining dormant in some other part of the body. As Hunt and Osman have pointed out, the tonsil may be simply the portal of entry in the first attack and later the organisms may be located in other tissues.

But should we not give heed to factors which may indeed be predisposing? Lowered resistance, poor environment, faulty hygiene, and incorrect ill balanced diet all may play a part, causing repeated flare-ups of a bacterial focus. In the majority of cases the period of activity of the infective organism in the tonsil is during the earlier years of life, when these predisposing factors assume more importance. This is admittedly so in the type of cases having local manifestations of tonsillar disease, while in the type suffering with systemic disorders, from a local infection in the tonsil, we may usually glean a history of throat trouble in childhood. Should we not ask ourselves the question more frequently, "Why does this child have infected or hypertrophied tonsils?" instead of simply satisfying ourselves that he or she has them? Perhaps we could not answer this, but it would be good for all concerned if we tried.

Much has been done recently along dietary lines. There is no doubt that an ill balanced or deficiency diet is productive of tonsillar disease during the period of life in which the lymphoid nodules are at their greatest activity. Clark's work on cats, investigating the relations of the size of the tonsil to diet, brings out the fact that diet is of the greatest importance in this regard. A high fat and high calories diet caused

a considerable increase in the size of the lymphoid nodules, both in the tonsil and in Peyer's patches. Dean has found that a diet deficient in the fat soluble A vitamines predisposes to paranasal sinus disease. Stucky, in his work among the Kentucky mountaineers, found that milk, eggs and butter were almost unknown as foods. Even the children rarely drank milk. Their diet consisted largely of fried foods, coffee, pork, corn, etc. He found these children suffering from tonsillar and adenoid trouble to a degree hitherto unseen in his experience. Those children, operated upon and their diet changed, adding milk, butter, eggs and cereals, showed almost miraculous improvement; while in cases where no dietary change was made, the operative results were very unsatisfactory. And it is not always the poor or underprivileged child alone who suffers from dietary error. Often the child of the socalled better classes is the victim of malnutrition. In our present day civilization a proper, well balanced diet is comparatively a rarity. And often the spoiled child of wealthy parents is as poorly fed as the poor Kentucky mountaineer or the inhabitant of the East Side slum. It isn't necessarily undernourishment that predisposes to lymphoid enlargement. An ill balanced diet may be the cause.

Only recently a woman, 40 years of age, consulted me in regard to her ears, complaining of some tinnitus and a feeling of fullness. Two years before I had carefully examined her, not that she had any trouble, but simply for a routine checkup, because she wished to be sure she was all right. Her husband was quite deaf and she naturally had her attention brought to this subject. My record shows that I had found nothing wrong. She had a slightly irregular septum, not obstructive; small tonsils, and no adenoids. The examination included inspection of the nasopharynx with the nasopharyngoscope. From this I feel certain that she had no excess of lymphoid tissue in her vault at that time. This last time she had a catarrhal involvement of both eustachian tubes. On examination I was surprised to find not only both tubal orifices swollen, but enough excessive lymphoid tissue in her vault to resemble a well defined adenoid. Her husband had been on a very rigid diet to reduce weight. She said that she had been following the same diet, although it was not what she herself

needed. All butter fats had been absolutely eliminated. She had had almost a continuous head cold, obstructed breathing and then this trouble with her ears. I feel certain that this condition was influenced at least, if not caused, by this faulty, ill balanced diet. Putting her back on a well balanced diet has shown improvement although it is too early yet to make any definite observation as to a complete return to the previous condition.*

We must also not overlook the all too prevalent fault of overeating. Clark's work, mentioned above, is of extreme significance. Highly milled grains, deprived of their natural vitamines, heavy starches, cane sugar products, and meat are apt to form too much of our dietary foundation, to the exclusion of milk, butter, eggs, green vegetables, fresh fruits, and whole grain cereals. The importance of the intestinal tract as a focus of infection which may manifest itself locally in the nose or throat has been brought out by the work of Dwyer and Connellan in investigating the significance of histamine in the stools. These stools all show a great degree of acidity, due to formic and oxalic acids, and a low colon bacilli content. Elimination of the sugars from the diet and alkalinization of the intestinal tract solve many a problem in which the throat may seem to be the offender. And yet, how rarely do we examine the stools in these cases of focal infection? While there is an enormous field yet to be investigated, the surface of which has only been scratched, we cannot overlook the fact that diet must play an important part in the etiology of many cases of tonsillar disease. As time goes on, we will think more of the prevention of these conditions in childhood by proper dietary and hygienic control, and less of the matter of surgical elimination.

The greatest cause for dissatisfaction with the tonsil operation, in my opinion, is due to hasty or incorrect diagnosis. Many an operation has been beautifully performed, but with little or no subsequent satisfaction to the patient, or the attending physician, because of this error. Too little attention is ordinarily paid to taking a careful and complete history. It is a great temptation to skip over this rather hurriedly and,

*Since the time this was written this patient has shown a return to normal.

perhaps unconsciously, lead the patient into giving us a story conforming to our already conceived ideas of the case, when a careful investigation into the patient's past might point to something radically different. This is especially true in cases of focal infection. Never should we forget that there are other possible sources of the trouble, quite as apt to be at fault as the tonsils. While a history of several attacks of sore throat is very suggestive we must always bear in mind that this may be only incidental and that some entirely different thing may be the cause of the patient's disability. Past and present history, as well as habits of living, should be most carefully scrutinized. The tonsils may show evidence of disease but the patient's complaint may be due to something quite different. Tonsillectomy in such a case may indeed be beneficial, but too frequently the patient, getting no relief from his trouble, is thoroughly dissatisfied.

A short time ago I had a patient referred to me to have a tonsillectomy, provided I found his tonsils diseased. His chief complaint was glossodynia. He had been in the hands of a number of different physicians and it had finally been suggested that his tonsils be removed. His tonsils were not normal; in fact I found quite a bit of debris in the crypts, but the history pointed to something more than a chronic tonsillitis. He gave a story of feeling fine for a time and then being very weak, tired and all in for a spell; a typical history of cycles of depression. His color was suggestive. I had a blood count made, which was reported as follows: white blood cells, 7,000; red blood cells, 1,220,000; hemoglobin, 70 per cent; differential polymorphonuclears 70, large mononuclears 20, small mononuclears 20, considerable poikylocytosis. The patient was referred back for further general examination, an enlarged spleen was found and a diagnosis made of pernicious anemia.* I cite this simply as an example of how easy it is to remove some structure which appears to be offending, when the cause of the trouble is something entirely different.

I recall another man who had been operated upon for tonsils and whose complaint was headache, which in his

*This patient died recently. Diagnosis confirmed.

case was due to a paranasal sinusitis. He had a high deviation of the septum to the affected side and a submucous resection relieved the pressure headache. Naturally he was disappointed in the result of his first operation. The importance of the teeth, sinuses and ears should never be lost sight of as possible foci of infection.

Barlow calls attention to the fact that oftentimes the failure of a tonsil and adenoid operation to effect a cure for the socalled chronic cold in children may be due to the presence of paranasal sinus disease which has been overlooked. Dean, some years ago, brought out the fact that this condition is much more common in children than generally supposed. It is too easy to condemn an accessible structure like the tonsil when the greatest offender may be elsewhere. There is an old saying that we must look beyond our nose. As diagnosticians we must see below the throat.

Of considerable significance in cases of focal infection is the finding of a history of sore throats in earlier life, but none during the past several years. Usually these people have had a focus of infection in the tonsil since childhood. Formerly they had a certain amount of resistance to the infecting organism, which resulted in a localized inflammatory process, manifested as a sore throat when there was a flare-up of activity. Nature raised a barrier to the passage of the infectious process. Gradually this resistance became worn down, local manifestations were less, there was little or no active defense, and, while the sore throats were less severe, or even absent, there was an ever increasing amount of infection invading the body, until finally we have a well established systemic disturbance.

While little is known concerning the relations of the tonsils to the endocrines, this matter should receive careful attention. The observations of Looper and others on the beneficial effect of tonsillectomy on many cases of thyroid disturbance is of significance. On the other hand many cases of endocrine pathology may be overlooked, and tonsillectomy prove far from satisfactory because of lack of consideration to the ductless glands. Careful metabolic study of many of these cases would be illuminating. We are all familiar

with the picture of the child with a pale, pasty complexion, unhealthy appetite, sluggish stools, chronic nasal discharge, more or less acne, and breathing through the mouth. There is a complaint of backwardness in school, or maybe eneuresis. Undoubtedly the removal of hypertrophied lymphoid tissue in the nasopharynx is beneficial in many of these cases, but should not attention likewise be paid to their general metabolism? The endocrines may be at fault, or an improper diet may be a factor, which, if overlooked, may deprive a perfectly performed operation of the desired successful result.

The conservation of hearing should be a matter of greatest importance to the physician. In many cases of otitis media, suppurative or catarrhal, the crux of the whole question of successful management lies in the removal of infected tonsils or adenoids. This should never be lost sight of, as the preservation of hearing and the prevention of untoward complications mean a great deal to the patient; and the elimination of pathologic lymphoid tissue from the throat may turn the tide. With more careful and intelligent attention being paid to the question of tonsils and adenoids we may well expect less chronic ear trouble, with defects of hearing, in the coming generation.

In all these obscure cases where we suspect that the tonsil is the cause of the trouble, we should always insist upon a careful general physical examination. We must never lose sight of the fact that the human body is one grand and glorious whole; that all its structures are more or less interrelated one with the other; although, in these days of rather intensive specialism, we are apt to overlook any part of the anatomy except that with which we are especially interested. Careful examination of the patient will, many times, bring out other possible causes of the trouble just as important, if not more so, as the tonsils. Here is where we need cooperation. We each have our own field of work in which we are more or less competent to make diagnosis. The utilization of the necessary consultants to assure the patient of the best available diagnostic ability is well worth while to all concerned. Each can add his contribution of information acquired and the patient will receive the benefit. The

internist, the pediatrician, the otolaryngologist, and such others as may be indicated, should all work together. None of these are sufficient alone. The physical findings of the internist on the throat are very apt to be misleading; while, I am free to say, I doubt if any otolaryngologist is competent to form much of an expert opinion concerning a cardiac or lung condition. If all cases were carefully and thoroughly examined by men competent in all indicated lines, before being subjected to operation, there would be fewer poor results. This examination should include the various forms of laboratory diagnosis, as indicated. The X-ray, blood and serologic tests, urinary and fecal examinations, as well as bacteriologic studies should not be neglected. Frequently cultural tests of the bacteria found within the tonsillar crypts are of the greatest assistance in determining whether or not the tonsil is the offender. Care to utilize every available diagnostic aid will eliminate many of the causes of dissatisfaction.

For the most part the ordinary inspection of the pharynx which passes for an examination is worthless to determine if the tonsils are infected or not. The obvious case will be diagnosed and condemned, but these large pendulous tonsils, with wide gaping crypts exuding debris, are often the ones least likely to cause trouble of a systemic sort. The size of a tonsil is of little importance unless obstructive to breathing. These large tonsils drain fairly well and are usually of pathologic significance only as to their local manifestations, as the cause of sore throats. The most important type of tonsil is usually overlooked. This is the small, buried tonsil, sometimes almost atrophic, with its crypts more or less sealed, and the products of infection draining into the system, via the lymphatics or the blood stream. A dull red hue of the pillars, especially the anterior, is of significance, while the presence of dilated blood vessels running over the face of the tonsil arouses our suspicions. Pressure on the anterior pillar often demonstrates products of infection. Palpation of the cervical glands is of considerable diagnostic importance, sometimes being the most important clue to a focus in a buried tonsil. Perhaps the most valuable means of ascertaining if an apparently innocent tonsil is infected

is by the use of suction. With a glass tube of appropriate size and a rubber bulb capable of giving fifteen pounds of negative pressure, we may be pretty certain to find any debris that may be within the crypts and can gain an idea as to the condition of the tonsil. Frequently we will be surprised by the size of the tonsil as revealed by the suction tube. Culture from the crypts may be of considerable aid. An idea of the activity of the tonsillar process may be gained by applying the suction tubes and as carefully as possible cleaning out the crypts, and then reapplying them again in forty-eight hours, noting the amount of debris. The tonsilloscope and tonsil illuminators all may furnish information. Sometimes a tonsil will appear quite innocent under every possible means of examination, and yet the history and general condition makes it very probable that it is at fault. And when examined under an anesthetic, the physician is surprised to see how much debris he can express from the crypts. I consider that examination under a general anesthetic is of great value in cases otherwise impossible to diagnose. Of course one is hardly justified in having an ether administered for this purpose alone, but oftentimes one may utilize an ether given for another purpose. In cases where there is an adenoid to be removed and the tonsil is suspicious, one can examine under the ether and then pursue the course indicated.

Assuming that the tonsil operation is properly indicated, the time of operating may be a factor in any subsequent unsatisfactory result. This frequently brings up a most difficult question, as to when to operate. Each case must be decided on its own merits, utilizing the same amount of common sense and surgical judgment as would be used in any other surgical problem. How young should we remove the tonsils? Or how old is it safe to operate? These are common questions. There can be no hard and fast rule. It all depends upon the condition of the patient and the pathology of the tonsils. We used to feel that it was unwise to remove tonsils before the age of three years. In the great majority of cases this still holds true; not so much because of the operative risk, but because the tonsil does not usually cause much trouble below this age. The period

of the tonsil's greatest activity is the period of the child's most active growth, from three years to puberty. At birth the tonsils are small and innocent. Rarely do they give trouble in these first two or three years of life. If they do, it is usually in a mechanical way, because their size may obstruct the child's breathing. If this be the case they can be removed, provided the child is a good operative risk, but it should be done quickly and skillfully, not subjecting the young patient to any more operative shock than is necessary. In case the child is not a good operative risk, it is the part of wisdom to postpone the operation until these conditions, contraindicating surgery, can be overcome. The idea, sometimes advanced, that operation in the very young is unsatisfactory and that the tonsils are apt to recur is unsound. The operation is more difficult, but with a little care the tonsils can be removed completely and without fear of recurrence. The same holds true for adenoids. The indications for adenoidectomy at this age are usually obstruction to breathing, otitis media, or a purulent rhinitis. In all these youthful cases, X-ray examination for the presence of enlarged thymus should be a routine.

In the elderly, if we can definitely satisfy ourselves that the tonsil is the cause of serious trouble and there are no contraindications to operation, we may be justified in advising it. But we should always ask ourselves the question, "Is it worth while to this individual, considering the age and expectancy of life? Will he derive enough benefit from it to make it advisable?" The importance of a careful general examination here can be easily appreciated and care should be used in the selection of an anesthetic.

Again in the patient disabled from some systemic disease, the time of operation assumes the greatest importance. Always the question of surgical risk must be answered. In tubercular cases operation is not considered advisable when there are any signs of activity in the lungs, and here is where we must depend upon the internist, trained in chest work. The same idea holds true for the cardiac cases. Operation during a febrile stage of an endocarditis is assuming great risk. We should wait until the activity of

the process has quieted down. Compensation should be established and the patient put in the best possible condition to stand the operation. Digitalization should be carried out and the operation undertaken only when the internist considers the risk worth taking. Every operative procedure and each anesthesia means a certain amount of shock. We should be fairly certain that the patient can weather this. While some men are advocating the removal of tonsils during an acute attack of tonsillitis, or of a peritonsillar abscess, I cannot agree with this, unless under exceptional circumstances. We all avoid, if in any way possible, operating in the face of any respiratory infection. What is tonsillitis, if not a respiratory infection? And can we for a minute think that the inflammation is entirely limited to the area of the tonsils alone? The cautious abdominal surgeon hesitates to remove an appendix in the presence of a coryza or any similar infection of the nasopharyngeal mucosa. Shouldn't the same caution be applied to surgery of the throat? I cannot help feeling that we are courting disaster when we attempt to operate at such a time. Porter advocates the removal of the tonsil during the acute stage in certain types of peritonsillar abscess, where the abscess is basilar and not easily evacuated. This seems good surgery, as the burrowing of the pus downward may be a grave source of danger to the life of the patient. In all these cases, if we only use the same surgical judgment that is employed elsewhere on the body, operating when the conditions, both locally and generally, are best suited for absence of complications and natural recovery, we will have better success and fewer excuses to offer afterwards. Sometimes it may be necessary to take a "long chance." The best available consultation may decide that the only chance the patient has is to get rid of the offending structures immediately. Then we are justified in proceeding, provided this is thoroughly understood by all concerned. But, as a rule, it will be well to wait for the most auspicious time. As physicians, all we can do is to assist Nature. We must be careful that we do not hinder or even defeat her endeavors by being a bit too hasty. "Fools rush in where angels fear to tread." And surgical caution is more to be valued than mere technic.

SUMMARY.

The popularity of the tonsil operation, and the too frequent and universal performance, is, in itself, the main cause of subsequent unsatisfactory results.

This is sometimes due to faulty operative technic, often at the hands of untrained operators and resulting in incomplete removal, disregard of other elements of Waldeyer's ring, postoperative distortion and untoward complications.

Hasty and incorrect diagnosis accounts for much dissatisfaction. Disregard of questions of hygiene or diet, inaccurate histories and insufficient general study of each case, with due attention to other possible sources of trouble, are frequently the causes of disappointment.

The time of operation is of greatest importance, especially in cases of severe systemic disability.

Careful study of each individual case with utilization of the best available consulting aid will remedy most of these difficulties.

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LXVIII.

A CASE OF ACUTE MASTOIDITIS ACCOMPANIED BY AN UNUSUAL FISTULA SYMPTOM.

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K. M., 43 years of age, a workman, after a severe attack of acute rhinitis, was attacked with a right sided acute otitis media. The pains in the ear were but slight—he consulted me only after his drum membrane had been ruptured spontaneously and a profuse purulent discharge appeared.

Status praesens: A tall, strong, well nourished man. The internal organs normal. Throat and the left ear normal. In the nose slight swelling of the lower turbinals; no discharge.

The right ear: Profuse purulent discharge, odorless, creamy in color. The external ear channel reddened, swollen; the drum membrane perforated—a small round shaped perforation in the posterior superior segment of the drum. The hearing diminished—whisper, 1.5 meter. Weber lateralized to the right side. Rinne negative. The right mastoid process slightly tender on pressure. The patient has some headache. Temperature 101° F., pulse rate 92.

After the application of a usual treatment the pain in the ear disappeared. In two weeks the ear was dry and the general condition normal. However, the small perforation in the drum still persisted. Three weeks later he called again with a profuse discharge from the right ear, tenderness and swelling over the right mastoid region and rise of temperature to 103° F. Pulse rate 82.

Two days later there appeared quite suddenly and simultaneously the most alarming symptoms of an acute involvement of the right labyrinth and especially of the right lateral semicircular canal. Vertigo, falling to the left side, nausea, vomiting, spontaneous nystagmus to the right side and paralysis of the right facial nerve. The fistula test was positive; compression nystagmus to the right; aspiration nystagmus to the left. The hearing diminished markedly—whisper ad con-

cham. Tuning forks: $C_{32} = O$. $C_{128} = +$. $C_{2048} = +$. $C_{4096} = +$. Rinne negative. Caloric reaction was not performed. Temperature 98° F. Pulse rate 74.

Next day, some hours before the intended operation, the patient told me that when fumbling with his fingers under the compress over the right mastoid he lost his equilibrium and became dizzy if he pressed upon one distinct spot behind the ear.

Examining the case thoroughly, I found as follows: On pressure upon a part of the mastoid process, about 3 cm. behind the fossa mastoidea and 2 cm. beneath, a marked dizziness, falling to the left and a typical rotary nystagmus to the right were provoked. However, these symptoms are not so well developed as when the usual fistula test is performed by means of a rubber bag. This particular spot is circumscribed, being approximately 1.5 square cm. large. The pressure upon any other section of the mastoid process is accompanied by no labyrinth symptoms, only some tenderness of slightly swollen soft parts is present. The tip of the mastoid is absolutely painless. I demonstrated the case to Dr. T. Meyer, who kindly assisted me at the operation and repeatedly tried to find out some other place in the right temporal bone from which this unusual fistula test could be provoked, but there was only this one little spot responsible for this symptom.

Operation.—A typical incision. The mastoid process is well developed, big. Cortex was laid free from the tip of the mastoid process to the linea temporalis and from the external bony meatus to the occipital bone. Not the smallest fistula was to be seen, even after the application of adrenalin solution and after thorough examination by means of a magnifying glass. The corticalis is very hard and sclerotic. Only in the depth of more than 1.5 cm. I found an accumulation of pus. The pus is odorless, creamy in color. Towards the antrum there were granulations and softened bone, which were carefully removed. The granulations and pus descend toward the tip of the mastoid, which was entirely removed. The lateral bony wall of the antrum was removed as when a radical mastoid operation is performed. The lateral semicircular was laid free, but even after the application of adrenalin solution no fistula was to be seen. The region of the facial nerve was very carefully wiped

out by means of a strip of gauze. The bony wound was smoothed by means of a curette and the wound partly sutured. Usual dressing.

Next day the condition of the patient improved markedly. All the labyrinth symptoms and facial palsy disappeared at once. The temperature was normal, pulse 78. The patient claimed that he heard much better. Four days later, change of dressing and removal of the sutures. The patient walks around—no dizziness, no nystagmus. The after treatment consisted in the change of the dressing every second day. In three weeks the ear was dry, the perforation of the drum closed. The hearing and the caloric reaction normal on both sides.

Epicrisis: The bacteriologic examination of the pus revealed streptococcus mucosus infection. It is well known that this particular streptococcus is responsible for a concealed course of acute mastoiditis and produces extensive destruction of the bone. The fistulæ in the bony capsule of the labyrinth, and especially in the lateral semicircular canal, are chiefly due to the action of two bacteria: tubercle bacillus in the chronic and streptococcus mucosus in the acute inflammations of the middle ear. From this point of view, the case described above is a typical one.

Quite unusual, and, so far as I know, not described as yet, is the fistula test in this case. The patient accidentally discovered that when pressing upon some distinct spot in the mastoid region there appear the same turbulent symptoms as if the usual fistula test by means of a rubber bag was performed. I expected to find a fistula in the mastoid process corresponding to the spot mentioned above, but not the smallest fistula was to be seen. Nevertheless, a tiny fistula in the bone communicating with the antrum undoubtedly was present, because no other logical explanation of the phenomenon is to be accepted. It is possible that the size of this fistula was so small that it could be discovered only by means of a microscopic examination of the bone. The author advises that this fistula symptom should be tried in every case when a usual fistula test gives a positive result.

LXIX.

WHAT SEPTA DEMAND OPERATION?*

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The technic of a septum operation is now practically standard, but what particular septum to operate upon is in dispute.

In prehistoric times the principal function of the nose was undoubtedly olfactory, as this sense was necessary for protection to the individual, but, through evolution, changes have gone on until now the principal function is respiratory.

In our modern way of living in overheated houses and with varying temperatures out of doors, this now principal function of modifying the air on its passage through the nose and to filter out dirt and germs has become intensified and important.

The ethmoid sinuses formerly contained turbinates covered with olfactory nerves. They are now only rudiments of these important structures, which drain and receive aeration, together with the frontal and maxillary sinuses, through the nasal cavity proper.

The nasal chambers are also really continuations of the eustachian tubes, so that the ventilation of the middle ear is practically through the nose.

The work of modifying the air is performed principally by the membrane covering the turbinates, therefore it is very essential that these structures be left in place. The septum is merely a fixed wall towards which the membrane covering the turbinal bones swells or contracts to enlarge or to narrow the nasal space as required.

The ideal nose, then, has a more or less thin, straight septum, the nasal cavities are equal in size, and the turbinates

*Read before the American Academy of Ophthalmology and Otolaryngology at Chicago, October 21, 1925.

are in place so that both sides are doing an equal amount of work in modifying the air on its passage through the nose.

Nasal septa are classified under three headings: normal, deflected, and thickened.

Deflected septa have no definite classification, because of their variety. The deflections may be in the cartilage or bone, or both up or down, forward or back, but as a rule they more or less follow the sutures. The socalled spurs are part of a deflection.

The thickened septum receives most consideration when the thickening is between the middle turbinates.

Deflected and thickened septa are the primary cause of changes within the nasal cavities.

Following a deflection in the septum, Nature, in trying to correct the spacing within the nose, thickens the membrane of the turbinates on the concave side and thins the membrane on the convex side, thus forming a compensatory hypertrophy of the membrane of the turbinates on the concave side and a compensatory atrophy on the convex side. On neither side is the membrane of the turbinates normal.

Under these conditions, the air, on its way through the nose, is not properly modified and leads to constitutional changes.

Patients become undernourished, thin, nervous and lacking in constitutional vigor.

A deflected septum or a septum thickened high up between the middle turbinates, besides causing changes in the membrane covering the turbinates, crowd the turbinates against the lateral wall and interfere with the drainage and aeration of the sinuses, subjecting the patient to a possible sinusitis.

These changes within the nose cause a more or less congested condition, making the membrane easily susceptible to infections, especially "colds."

If drainage interference continues long enough or is repeated often enough with the right infection, all the changes of sinusitis follow, from the acute to the chronic states with pus, polypi and attending symptoms.

A congested nose, either continuous or intermittent, causes a congestion of the membrane lining the eustachian tubes and leads to pathologic changes within the middle ear.

Assuming that an ideal nose is essential for free breathing, drainage and aeration, this alone is not a sufficient reason for operating on a septum which does not come up to this standard.

It is true that most people have a more or less deflected or thickened septum, with apparently no symptoms so far as they can judge.

No one can tell positively how much effect a not normal nose has on general constitutional conditions, but certainly the nose should be taken into consideration in all cases of so-called nervousness and in thin, undernourished patients.

I believe that nearly every case of sinusitis is due primarily to a deflected or thickened septum which interferes with drainage and aeration.

Certainly every case of repeated acute attacks of sinusitis—that is, a congestive sinusitis with pain, should have the septum carefully inspected as to a deflection or thickening in the region of the middle turbinates. Practically all chronic sinusitis cases with pus and polypi need a submucous resection together with the sinus operation if we expect to permanently benefit these patients.

These seem radical statements to make, but I believe one of the failures in the cure of sinus cases is due to the lack of this operation. Not properly studying the cases is the only reason I can give for not observing these points.

In cases of middle ear pathology, the nose must be free if we expect proper aeration through the eustachian tubes.

A complete submucous resection must be performed in every case, and particularly must the bone be well taken out between the middle turbinates.

It is surprising what poor submucous resections are performed even by our socalled good men, and it is no wonder they get poor results. This is probably not due to their lack of surgical ability, but rather to their surgical judgment.

My contention is that in too many of these cases the bone is not fully removed in the region of the middle turbinates and too much bone is left above or below, causing tenting of the membrane.

Instead of too many submucous operations not enough are being performed, if we study these cases properly and expect to get results.

But above all things perform a complete operation.

SUMMARY.

The following class of cases should be considered for septum operations to obtain proper modification of the air passing through the nose, and for drainage and aeration of the sinuses and eustachian tubes:

Socalled nervousness,
Thin and under nourished patients,
Mouth breathers,
Patients who have repeated colds, or "stuffy" noses,
Repeated acute congestive sinusitis,
Chronic sinusitis with pus and polypi,
Asthma,
Hay fever,
Middle ear pathology.

LXX.

"HORS D'OEUVRES."

By George H. Thompson, M. D.,

North Adams, Mass.

Case I.—J. B., age 6, never a strong child, was subject to fits of temper, when his parents had no control over him. He was sent to me for the removal of his tonsils and adenoids. The day following the operation he was taken home, and four days afterward his mother telephoned me, saying he seemed well and asking if he might sit on the piazza. Permission was given.

Ten days after the operation his father came to me and asked me to see the boy, as he was hoarse and not inclined to play or eat. While we were going to his place, which was ten miles out in the country, he told me that during the last two days the child had had two of his "temper fits," as he called them, and had yelled himself hoarse, which explained that symptom to me as well as to the father.

I found the child's temperature 99.8, pulse and respiration normal, and the upper part of the throat normal. Any attempt to examine his larynx with a mirror or spatula met with such violent opposition that the parents would not allow it, nor was I anxious to attempt it. There was a thin watery nasal discharge but no membrane in the nose.

I was not impressed with the serious illness of the child and ordered a sedative, not expecting to hear from him again. In twenty-four hours I was hurriedly called, to find a typical case of laryngeal diphtheria. Prompt intubation and large doses of antitoxin saved his life and greatly relieved my mind.

Upon inquiry, we could not find that there was any diphtheria in the neighborhood. This child also about a year before had had a positive Schick test and had had two doses of the toxi-antitoxin, but had not had the third dose. Where this child picked up this germ I do not know, but one thing I do know is that hereafter a child who is hoarse, whether subject to fits of temper or otherwise, will have his larynx examined.

Case II.—A. C., age 45, a farmer, while going out to his barn in the early morning of September 2, yawned and while doing so felt something go into his mouth. Before he could expel it he was conscious of a stinging sensation in the upper part of his throat. Clearing his throat by coughing and hawking, he expelled an ordinary bumblebee.

Shortly afterward he began to find it difficult to breathe, and the whole household became wildly excited. I was summoned and saw him at his home, twenty miles out in the country, about two hours after the advent of the bee. At that time he was gasping for breath and seemed about to expire.

A hastily performed tracheotomy, without any of the ordinary surgical precautions, proved a life saving measure. He made an uneventful recovery, although it was ten days before he could go without the tube, so intense was the swelling around the upper part of the larynx.

Tracheotomies in a small practice such as mine are infrequent, and sometimes those for which we plan and arrange all the details in advance are not so successful as this one was.

Case III.—E. M., age 22, while away on a vacation, had a cold and earache for two days. She consulted a doctor, who supposedly opened her drum membrane. The incision did not relieve her pain, and I saw her two days afterward. At that time her temperature was 101, pulse 90 and respiration 20. The drum membrane could not be seen, as the external auditory canal was so generally swollen. There was no discharge and no symptoms referable to mastoid. After a few days in the hospital, with irrigations and general medical treatment, she felt quite well and her temperature was normal. Her external auditory canal was still greatly reduced in caliber.

She was perfectly well for three weeks, when she told me that for the past two nights she had had a good deal of earache and pain over the side of her head. Her temperature was 100, pulse 90, respiration 24. That portion of the drum which could be seen was somewhat red and soggy. She was tender over the antrum, tip and emissary vein, and the X-ray indicated that the mastoid was extensively involved. That night I operated on her mastoid and found considerable breaking down, with pus in practically all the cells. The sinus was located far for-

ward and exposed through nearly its whole extent. It seemed normal in color and to palpation. The bone was thoroughly cleansed and she made a prompt and good recovery and was back at work in four weeks, apparently well. The only abnormality that was noted was that her ear canal remained very much reduced in size.

Five weeks after she was discharged she came in to the office on a holiday, stating that she was ill. Her temperature was 103, pulse 110, respiration 24. As there was some typhoid in her neighborhood, I thought she might be coming down with that disease, and I sent her to the hospital for observation and general medical care. That evening her temperature was 105, pulse 130. Her blood count showed a leucocytosis of 18,000, with 85 per cent of polymorphonuclears. Her chest, abdomen and kidneys and central nervous system were negative. During the following day her condition seemed much improved, but in the evening, following a chill, her temperature touched 105, with pulse 130, and she appeared desperately ill. We decided to expose her sinus without waiting for the results of the blood culture.

There was nothing in the mastoid to account for her general symptoms. The sinus was thoroughly exposed and was of a dirty gray color. The internal jugular was exposed and seemed to me to be normal in color and feel. It was ligated above the facial vein.

The sinus was then opened and a large, fairly firm thrombus removed. This was followed by very profuse bleeding from the torcular end, but scarcely any from the bulb terminal, nor was I able, after the removal of several small clots with the curette, to obtain as free bleeding. The cultures from the blood as well as from the thrombus showed numerous colonies of streptococcus hemolyticus. Unfortunately both the culture tube and the smear made at the time of the mastoid operation had been lost en route to the laboratory, so we do not know what microorganisms were then present.

There was severe shock from the operation and hemorrhage, and for several days the patient was extremely weak, her temperature ranging from 100 to 103, and pulse from 100 to 140, in addition to which she had to a marked degree meningeal

irritation, so frequently seen after jugular ligation. On the seventh day her temperature was 105.5. We opened the neck wound and irrigated it. This was followed by a lowering of the temperature and a gradual recovery. She left the hospital four weeks from the date of her admission.

So far as I can ascertain from reading, cases of sinus thrombosis which do not make themselves evident for at least twelve weeks after the onset of the original infection are not common.

The patient was then and is now my office assistant, and I followed her case with great interest and for days with anxiety.

SOCIETY PROCEEDINGS

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Regular Meeting of Monday, February 1, 1926.

THE PRESIDENT, DR. HARRY L. POLLOCK, IN THE CHAIR.

CLINICAL PROGRAM.

Congenital Bony Occlusion of the Posterior Choana.

BY JESSE H. ROTH, M. D.

A patient, aged 35, came to the dispensary complaining of complete nasal obstruction and profuse mucopurulent discharge from the left side of the nose, which had been present since infancy. She gave a typical history of syphilis, which had been contracted since marriage. She had received antisyphilitic treatment and when presented the Wassermann reaction was negative.

There was a history of a nasal operation in an attempt to remove the obstruction six years before.

Examination showed the left side of the nose filled with mucopus. The mucous membrane was pale and glossy. It was impossible to pass an applicator into the pharynx, the resistance to the applicator being firm above but not below. The postnasal mirror showed complete obstruction of the left choana. There was malformation of the palate, especially on the left. The arch was well formed and not unusually high. The left maxillary sinus had been washed repeatedly and mucopurulent secretion was always obtained. The ethmoids were also involved.

DISCUSSION.

DR. ROTH said they intended to investigate and see if the surgeon had removed the occlusion even with the floor. If not, they would use electrocoagulation and see if they could not get a good, smooth healing.

Atypical Falsetto Voice.

By Elmer L. Kenyon, M. D.

The patient was a young man, who gave a history of having a disturbed voice at seven years. The classification had not been worked out. The concavity of the cords was attributed to paresis, either of the cricothyroid or the thyroarytenoids, with a general disturbance involving the extrinsic musculature, which originates much more often in the mental act of voice production than in local physical or functional disturbance. When the patient talked, the thyroid cartilage plunged quickly to the highest point attainable and stayed there quite constantly, under tension. The extrinsic muscles at the base of the tongue were likewise tense, making normal approximation of the vocal cords utterly impossible. Dr. Kenyon believed the disorder to be due to psychologic confusions with respect to voice production.

DISCUSSION.

Dr. J. Holinger stated that in singing the complete relaxation of the muscles of the pharynx is very important, and singing teachers long ago resorted to having the patient hum, or sing with the mouth closed. He thought this might help in Dr. Kenyon's case. If one taught the boy to hum, at the same time watching the neck and instructing him to relax completely, it might overcome the tenseness of the musculature.

Dr. Kenyon (closing) called attention to the cases reported by Dr. Makuen a number of years ago, which were of much the same high pitched type as the one presented. In one of them the patient was instructed to talk in a low pitched voice, which he did. Following this he did not return, and the Doctor's supposition was that he ever after talked with a normal voice. Dr. Kenyon believed that in the attempt that has been made to cure the high pitched voice in the past, errors have been made in thinking that the technic had been corrected. By using the imperfect technic of these individuals they can lower the pitch considerably. The ear may be unable to decide whether the patient is using the normal or abnormal technic unless one studies the extrinsic musculature. He felt practically sure that much more definite measures must be taken

than those suggested by Dr. Holinger in order to secure the proper voice. He teaches the individual what his voice really is, then what the tone ought to be, and then how the extrinsic musculature acts normally. After the patient has learned to produce voice with a normally acting extrinsic musculature there is comparatively no difficulty and the result invariably is a normal voice.

Complications of Ear, Nose and Throat Operations and Diseases.

By Joseph C. Beck, M. D.

Case 1.—The patient was a woman, who was presented because of the numerous operations she had undergone for the cure of mastoid infection. She had had five operations in a period of seven years. She gave a history of recurring attacks of dizziness, vertigo, headache and swelling in the region of the neck. There was nothing unusual in the examination of the canal except evidences of modifications of the radical mastoid operation and pus from the posterior part of the operated mastoid region. The ear stood out, and the posterior part of the concha was very thick, as if it might contain an abscess. This condition of the concha had existed for two years. There was practically complete deafness present. The most interesting part of the case was the roentgenographic examination, which disclosed a large pneumaticized area with a large number of cells. A stereoscopic roentgenogram of the operated ear showed a number of cavities which might be interpreted as due to the previous operations or to unopened mastoid cells. Dr. Beck had recently operated on this patient. He had made an auricular incision at its junction with the mastoid process and another close to the hair line, bringing down a flap and exposing the whole mastoid area. This disclosed a large number of cavities which had been obscured and which proved to be unopened mastoid cells which perhaps had been missed at the previous operations. Since this operation the patient had been free from complaints.

Case 2.—This patient was a man, who first consulted Dr. Beck on January 4, 1926, because of defective hearing, soreness and ringing in the left ear, which had been present for several months. There was no history of acute infection of the ear. There was frequent retention of wax in the ear, and at

this time removal of the wax caused some soreness and swelling in the canal. There was almost complete stenosis and redness of the canal; not much involvement of the auricle. The hearing tests showed more reduction in the upper tone limit (audiograms presented). The patient's occupation was making steel pipes, during which there was a great deal of noise and shaking. The diagnosis of nerve deafness was made. Examination of the canal showed an exostosis, situated at about the center of the canal, which practically shut off the inner portion of the external canal. The roentgenogram showed the outline of the exostosis but no evidence of clouding of the mastoid cells. He expected to operate the following morning and clear the canal of the exostosis by means of a retroauricular incision.

Case 3.—A man, of 43, entered the North Chicago Hospital with a history of having an acute edema of the larynx after being out hunting. He had had similar attacks before and had been hoarse for a number of years. He was in good health and had no complaint except loss of voice and difficulty in breathing during these attacks. Examination of the larynx showed a unilateral swelling on one side of the right cord; it appeared to be smooth and confined to one side. There were no palpable glands in the neck. The man was very plethoric and drank and smoked moderately. The blood Wassermann reaction was negative. Physical examination showed no evidence of any involvement of the lungs. A diagnosis of the neoplasm was attempted by suspension laryngoscopy but they were unable to expose the larynx. They then resorted to the indirect method, but by no manner of means could they get near the larynx. They finally secured a piece, but not of sufficient size to permit of diagnosis. Following this procedure the patient developed great difficulty in breathing, and a tracheotomy was done the following day with difficulty. In doing this no more than one and a half inches of trachea could be exposed on account of the patient's fat neck. The muscles of the neck were very large, and a large thyroid gland was encountered. After placing a large tracheal tube in the opening they had to pack it to stop the oozing. The following day the man developed an infection around the tracheotomy which was very unpleasant. No definite diagnosis had been possible be-

cause of inability to secure tissue for biopsy, but this will follow.

Case 4.—This patient had had a radical mastoid operation several years before, which had nothing to do with his present illness. He came to the hospital with a boil on the nose, with edema of the eyes and a septic temperature. There was a hard lump over the cheek but no evidence of sinus involvement. No incision or manipulation had been attempted, but the patient had been treated with a hot light over the face. This had given uniformly good results in similar cases.

These four cases were shown, as they illustrated the complications that arise in both nose, throat and ear diseases as well as operations.

DISCUSSION.

DR. G. W. BOOT, discussing the patient with the multiple mastoid operations, mentioned a patient who was a nurse, who had had seven mastoid operations and wished to be operated on for a brain abscess, but there was no sign of such a process. The patient had been sent to the country for a rest from hospitals and doctors.

In the tracheotomy cases he thought a sharp hook indispensable.

DR. SAMUEL SALINGER asked for an explanation of the edema or hyperplasia behind the ear in the mastoid case.

DR. C. H. LONG thought the mastoid case was important because Dr. Beck found the cells not previously opened by the other operators. He believed one should be more careful in searching out the mastoid cells in an effort to get successful results, and that thorough roentgenographic examination should always be made.

DR. G. H. MUNDT (by invitation), discussing the case of exostosis, said he was reminded of the case of a physician who came in with a history of not being able to hear in one ear with a stethoscope. Examination revealed swelling of the posterior superior wall in both ears. He made a diagnosis of exostosis, but advised the patient to have this confirmed by another otologist. This specialist made the same diagnosis, and, as the patient wished to be operated on, he was referred to Dr. Beck, feeling that the exostosis was too near the middle

ear to remove it with safety. Dr. Mundt asked Dr. Beck to tell of his experience with this case.

DR. BECK (closing) agreed with Dr. Boot regarding the use of a hook in the tracheotomy cases and said the difficulty in the case he reported was to get down to where one could put the hook in. This could not be done because of the large amount of thyroid that was present.

He thought the cases with numerous mastoid operations were not uncommon, but that Dr. Long's point regarding the value of the roentgen examination was good. In his case, when the tops of the cells which had not been opened in the sinus were removed, the cells were bluish and proved to be large, infected mastoid cells, a chronic infection of the lining membrane of the cells. He was not sure whether the results would be permanently good or not, for it was too soon after operation. Some types of cholesteatoma do not respond to any operation. The consistency of the swelling behind the ear was not that of cartilage but of indurated inflammatory tissue. There was no marked thickness of the cartilage of the auricle. By taking a separate picture of the cartilage they were able to differentiate it from inflammatory tissue.

In the case of nerve deafness the patient had been given potassium iodid in small doses. The question was whether previous local treatment had had anything to do with it. This was an important question for this patient, for he claimed that following treatment of the external ear he became deaf. If this was merely nerve deafness, which had disappeared, it was a good point for his attorneys to fight over.

Dr. Beck said that many of the cases with exostosis occur with fistulæ, and Dr. Mundt's patient had an exostosis which bothered him a great deal. He attempted to remove it but it was not located as well as in the case presented. It extended down to the middle ear, and he did not remove any of it for fear of injuring the facial nerve.

The man, who evidently had a carcinoma, was now ready for a laryngectomy, but from what they had learned during the tracheotomy they would not do a laryngectomy, either from above down or from below up, but would do a laryngofissure as soon as the condition was completely cleared up. This would be done by means of the new apparatus for electro-

coagulation (radio knife), which would cut through all the tissues and seal them up against bleeding and implantation as it went along, for it cuts through cartilage as well as through soft tissues.

Sinusitis With Unusual Polyp Formation.

By C. H. Long, M. D.

The patient was a man, aged 37, who had had infection of the nasal sinuses for many years. This was thought to be so-called catarrh, and no attention was paid to it until he was accidentally hit over the ear by a fellow workman, which resulted in an acute mastoiditis. When he sought medical aid for this condition he was informed that his nose was filled with polyps and that they were probably the cause of the condition of the ear. The polyps were removed but the ear condition grew worse. Another physician was consulted, and Dr. Long was called in consultation. A diagnosis of mastoiditis was made and a simple mastoid operation was performed. Following healing of this wound the nose was examined and many polyps were found. A diagnosis of chronic double maxillary and ethmoid sinusitis was made, complicated by a badly deflected septum. At the time of this operation it was discovered that both sphenoids and the right frontal were also involved and these were cared for later. At the time of presentation both of the maxillary sinuses were practically cleared up and apparently all the ethmoid cells had been evacuated and curetted. The anterior sphenoid walls had been punched out two or three times, but there was a tendency to reformation of the polyps.

Unusual Mastoid Region Tumor.

By H. L. Pollock, M. D.

Case 1.—The patient was a man who had a growth of about two years' duration behind the left ear. There was a history of its increasing and decreasing in size. When first seen, about three weeks previously, the mass was about the size of a small egg and apparently fluctuated. There was no rise in temperature and no pain. Roentgenographic examination showed a cholesteatoma of the right ear, but no bony cranial defect on

the left side. An incision was made very carefully under synergistic anesthesia, and they came down upon the growth, which seemed to be dural. After opening the lower part, Dr. Pollock explored the inside with a finger and felt something like a tooth. Upon careful dissection the tumor came away without difficulty and the wound healed by primary intention. Microscopic examination showed the growth to be a lipoma with calcareous degeneration.

Carcinoma of the Larynx.

By H. L. Pollock, M. D.

Dr. Pollock presented a specimen from a case of laryngectomy and photographs showing the condition of the patient. The patient was a merchant, aged 67, who had begun to get hoarse about two years previously. He consulted his local physician, who treated him for six months without examining the larynx. He then consulted a specialist, who advised the use of radium, and this had been used about a year previously. When the man consulted Dr. Pollock the right side of the neck showed scarring from radium burns, but apparently the treatment had helped him some, for his voice cleared up and no metastatic glands could be palpated. The same physician had advised more radium six months before, but the patient would not submit to further treatment. Examination revealed an irregular growth on the left side of the throat. Under suspension laryngoscopy a piece of tissue was removed, which proved microscopically to be squamous cell carcinoma. The patient was given the choice of three things: first, to let the condition alone; second, a tracheotomy; third, a laryngectomy. The latter was decided upon. A two-step operation was performed. A tracheotomy was performed about the middle of December, and about three days later the patient returned home, coming back on January 11th for the laryngectomy. He had had no trouble with the tracheotomy but was kept under observation for three days before the laryngectomy was performed, on January 14th. It was necessary to change the entire method of operating. An incision was made which began at the angle of each jaw and continued to a point below the tracheotomy opening, and a flap was raised, which gave a good exposure. Dr. Pollock then started at the top and dissected

off the larynx first. This offered great advantages. The patient was operated on under synergistic anesthesia and was conscious all the time. Very little local anesthesia was used. In dissecting downward he dissected the mucous membrane as he went along and had much more mucous membrane than usual. The second advantage of this method was that no blood ran down the trachea. The tracheotomy tube was kept in place until the larynx was entirely dissected; it was then removed and a long rubber tube was used to plug the trachea up tight so that nothing could seep into the trachea from the wound. The opening was larger than usual, which was thought to be due to pressure from the tube that was placed in the trachea. The patient was making an excellent recovery and was up and about the hospital and grounds.

Some of the unpleasant things in laryngectomy are due to inhalation of material from the wound. In this case the tube and packing were kept in for eight days following the operation. The patient had been fed by an esophageal tube and had only a small pharyngeal fistula. On the right side, where the large amount of radium had been applied, it was almost impossible to dissect the tissue out, while on the left side the dissection was easy. There had been no untoward result from the operation, and because of the method used there had been much more mucous membrane than usual to use in closing the esophagus.

Report of a Case of Streptococcus Infection; Peritonsillar Abscess.

By G. W. Boot, M. D.

The patient was a well developed boy, aged 8, whom Dr. Boot had been called to see and to open a peritonsillar abscess on the right side. There was so much swelling that no anesthetic could be given. An incision was made with a blunt tonsil knife and extended through the mucous membrane and fascia only. No pus was obtained. The patient had previously had a discharge of pus from the nose; he also had an abscess in the left ear which Dr. Boot opened. The temperature was 105° F. He improved steadily for about a week. The temperature returned to normal, the swelling disappeared from the peritonsillar region, but the left ear continued to discharge

bloody pus. Dr. Boot was considering the advisability of a mastoidectomy, when one morning at 4:30 he was called by the nurse, who said the boy had had a severe hemorrhage of probably two quarts of blood. He went to him immediately, but found the hemorrhage had ceased and there was no evidence of where it had come from. He was given salt solution by enema and ceanothen by mouth. He was seen again at 8:30 and was semicomatose; he was ordered to the hospital for transfusion, and a general surgeon gave 400 cc. of Ringer's solution under the breast and 100 cc. of the father's blood intramuscularly. Following this the boy's condition improved and he slept most of the day. At 2:45 p. m. he was in good condition, but within five minutes had another hemorrhage and died. No postmortem examination was permitted, but by holding his mouth open and the palate up as well as possible with a blunt hook Dr. Boot could see an enormous vessel, as large as a lead pencil, running up behind the posterior pillar on the right. In the nasopharynx this vessel had sloughed through in two places, at least an inch above where the incision had been made for the peritonsillar abscess a week before. It was impossible to tell whether the vessel was an artery or a vein, but it appeared to be an artery. It lay just under the mucous membrane and just behind the posterior pillar.

DISCUSSION.

Dr. C. H. Long thought it would not have been a bad plan if Dr. Boot had ligated the common carotid artery.

Dr. J. Holinger recalled the patient he had presented before the society a number of years ago, who had parallel to one posterior pillar a large vertical pulsating blood vessel which was easily visible. Had there been a hemorrhage in that case there would have been no possibility of getting an aneurysm needle around it or any way of getting to it. Since then he had read reports of similar findings. Consequently he thought such vessels occurred not infrequently and were very dangerous.

Dr. Samuel Salinger said that about a year ago, at the meeting of the Royal Society of London, this question was discussed, and it was the consensus that this vessel was a tortuous internal carotid artery, which would remain so. The

recommendation was to palpate this region of the pharynx preceding every tonsillectomy.

DR. H. L. POLLOCK recalled a similar case, which he had presented, in which he anesthetized the patient's throat and with a small stethoscope could hear the bruit in the vessel. The general belief was that this was an anomalous internal artery, and he believed all these cases were considered to be the internal carotid artery.

DR. BOOT (closing) stated that during life there was no apparent source of the hemorrhage, and no opportunity to ligate the internal carotid artery. He asked Dr. Pollock whether he had secured a section from the tumor in the case he presented, and if there was a possibility of its being a mixed parotid tumor.

DR. POLLOCK (replying to Dr. Boot) said that they first suspected a dermoid cyst, for it felt as if it contained a tooth, but microscopic section showed it to be a pure lipoma with calcareous degeneration. The patient had a cholesteatoma in the opposite ear, but this had nothing to do with the ear which had the tumor.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Regular Meeting of Monday, January 4, 1926.

THE PRESIDENT, DR. HARRY L. POLLOCK, IN THE CHAIR.

Ephedrin.

GEORGE F. FISKE, M. D., presented the following preliminary report on ephedrin:

"Most of you have seen Dr. George Fetterolf's report upon ephedrin sulphate in the August number of the Archives of Oto-Laryngology. Dr. Fetterolf was so kind as to help me to obtain a small supply of both the chloride and the sulphate, and this is a short report of its effects upon the nasal mucous membrane.

"It was used upon twelve individuals, including myself. The solutions were 2 per cent, 1 per cent and 1 part to 500. The most satisfactory and least irritating method of application seems to be a cotton pledget rolled and saturated with the solution and then introduced into the lower meatus and left for two or three minutes in contact with the mucosa. With the 1 and 2 per cent solutions the shrinkage began immediately and was very complete within two to four minutes. The contraction remained complete for from one-half to two hours, the relaxation coming on more quickly in cases of acute rhinitis. The solution, 1 part to 500, caused a marked shrinkage, but not complete enough for operative purposes. In several cases the 2 per cent solution was used in connection with a 4 per cent solution of cocain, both separately and with a mixture half and half of the two solutions, producing the effect of both. No irritation was observed and no after effects, either good or bad. The action as regards shrinkage of the mucosa was very like the 1 to 1,000 solution of epinephrin chloride, except that the paleness produced was less marked.

"The aqueous solution seems to resemble the sulphate of atropin solution in its stability, which is of marked advantage. Its field of usefulness would seem to lie in intranasal operat-

ing. The alkaloid itself I have not obtained as yet, though I hope to. Both the chloride and the sulphate crystals are easily soluble in water. The price quoted me from Philadelphia was $1.50 per gram. The supply at present is limited, which is certainly understandable, if the plant ephedra vulgaris must be brought from China.

"I had hoped to have a larger supply in order to give it to all members of this society who are interested, but though I have ordered some from Dr. Read in Peking and also from France, it has not yet arrived. When it does I shall be glad to divide with you. It will then be tested with novocain for local anesthesia. While the expense at present is very great, there are some of us who remember paying one dollar a grain for cocain."

DISCUSSION.

DR. JOSEPH C. BECK said that Dr. Fiske was kind enough to let him have two small bottles of the solution, but after using it for two or three weeks, Dr. Fiske wrote him that the solution had not the strength that he thought it had, for it was less than 2 per cent. Dr. Beck employed it in application as well as spray form, and in this weak solution there was some shrinkage of the nasal mucosa. He had not observed any reaction, such as sneezing, which occurs following the use of adrenalin and epinephrin. He believed it would be a satisfactory solution when perfected and an aid to therapeusis.

DR. OTTO STEIN thanked Dr. Fiske for the supply of the solution which he had placed at his disposal. He had applied it with a dropper, placing two or three drops of a 1 per cent solution in the nostrils, with same effect Dr. Fiske mentioned. There had been no complaint on the part of the patients. He found the contraction very suitable with two drops in each nostril and thought it ample for all purposes, used either with a weak solution of cocain or alone. He had not studied the lasting effect but had used it routinely in most of his cases. The contraction occurred immediately and evidently remained for some time. He considered ephedrin a wonderful addition to the therapeusis for local application, and could not see the need of a 5 per cent solution when a 1 per cent acted so satisfactorily.

DR. A. A. HAYDEN stated that he had used ephedrin in a 1 per cent solution and found the contraction and blanching of the mucous membrane was quite as prompt as with the oily solution 1:1,000 of adrenalin. He had not used it in operative work but in the office. He tried it in his own nose and thought the contraction lasted for two or three hours. The advantages of the solution he believed to be as stated by Dr. Fiske and considered it of great value.

DR. FRANK NOVAK, JR., asked if anything was known concerning the toxicity of the drug.

DR. JOSEPH C. BECK said that Dr. Fetterolf of Philadelphia told him the laboratory was working this out at present and that information would soon be available.

DR. GEORGE F. FISKE said that a report in the Journal, by a Dr. Chang, stated that it had been used in shock and that there was no toxicity at all.

Congenital Osseous Atresia of the Posterior Nares; Case Report.

BY JESSE H. ROTH, M. D., Kankakee.

(ABSTRACT.)

The case reported was that of a girl, aged 12, who was slightly anemic, and gave a history of never having been able to breathe through the right side of the nose. Removal of adenoids in early childhood had given no relief. She had never been able to clear the right side of the nose of mucus, which at times caused a slight eczematous condition about the ala. There was no asymmetry of the face, and the palate was only slightly higher than normal. Examination showed the right side of the nose filled with glairy white mucus, which was removed by lavage. The turbinates were slightly atrophic and the mucous membrane paler than on the left side of the nose. It was impossible to use a postnasal mirror, for a complete bony obstruction filled the posterior part of the choana, probably situated at the posterior border of the hard palate. The plate extended from the floor of the nose and the septum outward to the lateral wall and backwards to the sphenoid. About midway up and close to the septum there was a small canal that would admit a probe. This canal extended toward the septum but ended in a blind pocket. Firm resistance was met in every

part of the obstruction. The parents refused to submit the child to operation and further investigation was impossible.

DISCUSSION.

DR. W. E. GROVE (Milwaukee) thought the difficulties in handling these cases were not the operative procedures, although these were sometimes difficult because of the age of the patient, but the difficulty in keeping the opening from closing. Many different methods of doing this have been suggested. He mentioned an interesting article which appeared in the Acta several months ago in which the authors, Bourgeois and Poyet, advised making the opening through the atresia by whatever method one wished and then allowing the secondary membrane to close the opening in again, which it will do almost completely if left alone. After this had taken place they then employed electrocoagulation of the membrane and reported some excellent results. Dr. Grove had operated on a patient of this sort, a woman, aged 42, who consulted him because of difficulty in hearing. He had no trouble in keeping the opening patent and made his approach through the posterior portion of the septum.

While studying his case it occurred to him that a nose which was obstructed in this fashion would be ideal ground for sinus infection, and on making roentgenograms he found practically all the sinuses on that side of the head were much blurred, but on opening the antrum found only the presence of a heavy, glairy mucus. Dean had called attention to the presence of sinus infection in all the children he had seen with congenital bony obstruction of the choana.

Vogel had recently called attention to the coexistence of this condition with various congenital anomalies of the face, such as fistulæ, malformed alæ, unilateral sweating and the like.

DR. J. HOLINGER said the occlusion of the choanæ was more a question of degree than of straight pathology, as all transitions exist, from wide open to a very narrow and to the completely closed choana. The closed choana gives the most symptoms, but often mouth breathing is due to a congenitally narrowed choana. He thought the small choana was more frequent than is generally diagnosed or suspected. He knew of

four cases of complete occlusion of both choana. He said in newborn or small babies he would hesitate before he filled the nose with water, for if there is a small opening the water is likely to run down into the windpipe and make trouble. A safer means of diagnosis is the use of the Politzer method, without closing the other nostril. The child will cry, and under normal conditions the air and mucus will come out of the open nostril. If this does not occur there is indication of occlusion.

He agreed with Dr. Roth and Dr. Grove that it is very difficult to keep the opening in the choana patulous after operation. Such a case must be watched for a long time. As soon as granulations form they must be cauterized. As a rule, persistent treatment will establish a permanent opening.

Dr. Elmer L. Kenyon said that he treated such a case several years ago in a girl of 16 or 17. The closure was on the left side, and she had a decidedly deflected septum, with a marked curvature toward the opposite side. He opened the bony wall with a trephine and she breathed comfortably. He enlarged the opening with a burr a few weeks afterward and did not know the subsequent history of the case.

Dr. Austin A. Hayden thought there was always a considerable amount of danger present in attempting to force water or air into a nostril which is suspected of being closed at the posterior portion. The air, water or mucus can be forced out through the tear sac and a diagnosis made in that way, but at the same time the eustachian tube is quite as apt to receive the forced fluid, and this is likely to set up an acute otitis. He believed the inability to pass a catheter or tube from the nose back into the antrum would answer just as well as a means of diagnosis. He had never seen complete occlusion even of one side, or at least had not recognized it. It might have been that some of the "blue babies" who had died at the hospital had died from this cause. It seemed to him that it might be well for otolaryngologists to interest themselves in this question, with the idea of securing some postmortem examinations on these children, or at least a digital examination of the nasopharynx of any child who dies of such a respiratory disorder. The determination of the degree of patency seemed to him very important. In looking at the front of the nose

the septum and turbinates, if deformed, often are straightened out or removed, but this does no good if there is a narrowing behind, and the patient will still have the symptoms for which he sought relief. Dr. Hayden had seen quite a number of posterior openings that differed greatly in size on either side, and not a few cases of obstructed breathing had been explained to him and to the patient in that way, so that the patient would understand why the operation was not attended with all the good results they had anticipated.

Dr. Joseph C. Beck called attention to the paper of Dr. Lebensohn of two years ago and said that at that time in the discussion he mentioned their experience in a few cases from the diagnostic and therapeutic point of view. The roentgenogram had not been mentioned by anyone in the present discussion, but it is of great assistance in the diagnosis. In the treatment everyone has the same difficulty in keeping the opening patent because of the cicatrization, but in one case in a 7-year-old boy good results were obtained by passing a catheter through the nose after the opening was established and then bringing back a metal tube which was held in front and tied by means of a string through the opposite nostril. This was kept in place until complete epitheliazation occurred (about six weeks) and the opening remained patent. Dr. Beck thought it was not essential for a child to be old enough to permit putting the finger in the postnasal space, for any instrument held against the posterior wall of the pharynx similar to an adenoid curet, only solid, would enable one to use a bur without injury to the posterior wall of the pharynx.

Dr. C. F. Yerger asked whether the nasopharyngoscope would not be of advantage in these cases. He thought it could be used through the opposite nostril with good results.

Dr. Jesse H. Roth (closing) stated that since his paper was written three or four reports had appeared in the literature. They now had another patient under observation who had had an operation performed for congenital osseous atresia, but the opening had closed over. In that case the roentgenogram showed that the closure was intact. He knew of no means to facilitate the diagnosis aside from those that had been reported, and filling the nose with water was one of the means of diagnosis recommended.

Paper: The Theories of Hearing.

By J. Holinger, M. D.

(Abstract)

The theories of hearing are neither so clear nor do they explain all the phenomena so well that we should ignore all attempts at anything better. In the Annals of Otology, Rhinology and Laryngology of October, 1925, Specht described the great changes that the study of the soundbox has brought about. Edison used it in the phonograph and in the radio. Its laws must be valid in the middle ear. The sound waves produce changes of pressure in the middle ear, which are transmitted by the membrane of the round window to the labyrinthine fluid. The stirrup and ossicles act as a brace. The changes of pressure in the fluid become the irritants for the hearing cells.

Criticisms: Specht thinks that in hearing by bone conduction the vibrations of the bone are transformed directly into changes of pressure in the fluid of the labyrinth. He does not recognize the physical impossibility of that. He furthermore overlooked the works of Liebenmann's assistants, who proved that Corti's membrane has a very important function in the process of hearing. Finally, why should the drumhead and the chain of ossicles act as a brace? They are under the same changes of pressure as the membrane of the round window. Dr. Holinger thought it quite feasible to accept Specht's theory and combine it with Bezold's theory of the function of the chain of ossicles.

DISCUSSION.

Dr. Alfred Lewy said that according to his understanding of Dr. Holinger's presentation of Specht's theory it resembled very much the theory of Pohlman of St. Louis, which was, briefly, that the drum menbrane and chain of ossicles acted as a damper on the sound waves which entered both by air and by bone. Recently Fowler published an article on bone conduction in which he called attention to the fact that there is no such thing as purely air conduction or purely bone conduction. An impulse given to the bones of the skull is also partially

transmitted through the air in the external canal via the middle ear to the fluid of the labyrinth as well as directly through the bone, while impulses arriving through the air are also partially carried through the bone, so that the terms air conduction and bone conduction are really relative terms.

The question raised as to how the sounds transmitted through the drum membrane and middle ear can enter at the oval window to the exclusion of air vibrations transmitted directly across the tympanic cavity to the membrane of the round window was perhaps best answered by Bezold's investigations (quoted by Edelman). The area of the membrana tympani multiplied by its excursion distance is to the product of the area of the footplate of the stapes multiplied by its excursion distance as 778 is to 1; therefore, impulses transmitted from the membrana tympani through the leverage system of the chain of ossicles are increased 778 times in intensity when delivered to the fluid of the labyrinth at the oval window. Whatever air waves reach the membrane of the round window across the tympanic cavity are not increased in intensity by any such leverage system and must be very low in power compared with that exerted at the oval window. It thus seemed that Bezold's theory that the elasticity of the round window membrane merely permits movement of the nonelastic fluid of the labyrinth under the impulses delivered at the oval window. Edelman also called attention to the fact that the relation of the specific gravity of water to that of air is 774 to 1, approximately the leverage relation of the movement of the membrana tympani to the movement transmitted at the oval window, and that it is very improbable that this significant relation is a mere coincidence.

Dr. Robert Sonnenschein said that the ratio of 700 to 1 was beautifully shown in a series of articles collected by Edelman, so that one could see that the ratio of the drum membrane compared with the stapes. It seemed to Dr. Sonnenschein that one of the principal objections to the acceptance of the theory of Specht was that the oval window and the drum membrane, with the stapes, act merely as a brace. When a number of sounds are produced only one curve results, because a membrane can only react in one direction at a time. It is well known that the low tones have a marked amplitude of

excursion, and very little intensity, and it takes the lever action of the ossicular chain to transform these into tones of smaller amplitude, but greater intensity to permit them to be transmitted via the oval window. If there is loss of the proper mobility of the ossicular chain the low tones are not well heard. He believed that Bezold's original theory still held.

Dr. Frank Novak, Jr., expressed his interest in the paper but found that he had some difficulty in following the essayist. This was probably due to the difficulty of keeping clearly in mind the basic physics of sound. The essayist spoke of the disturbance in the air and then the changes in the endolymph, and mentioned the fact that the waves became pressure waves in a fluid medium. This was a little confusing. Is not sound simply matter in rapid rhythmic motion? Whether this matter be in a gaseous, fluid or solid state does not change the kind of energy. The type of energy is no different, whether it be present in a fluid or in a gaseous medium. Another point which interested him was the effect of pressure on colloids, and he asked the essayist whether his statements were supported by any experimental work on colloids.

Dr. J. Holinger (closing) said there had been experiments that showed that any outside change of the cell has an influence on the concentration of colloids in the presence of ions. This is Specht's hypothesis, but Dr. Holinger did not agree with this, in view of Liebenmann's experiments. Specht had not mentioned these experiments of overirritating the labyrinth of birds. Yoshee, in his atlas, showed that the hearing cells farthest from the pivot of the membrana tectoria were the ones which were most destroyed, while the hearing cells close to the pivot were very little injured or remained intact.

As to the lever action of the chain of ossicles, this was the point he brought against the theory of Specht. He believed the footplate of the stirrup did not act simply as a brace but that it has an active function, and the form of the joint between the mallet and incus proves this. The formation of the joint of the incus with the little tooth on the side makes it impossible for the hammer to move inward without taking the anvil along, while the hammer can move outward without taking the anvil along. If the ossicles acted as a brace this mechanism

could not be explained, while it fits accurately in the idea of the lever apparatus.

Dr. Holinger believed there was some good in the theory of Specht. Some points of it had to be changed considerably. Specht admits that Bezold was the most conscientious worker on the matter.

Dr. Robert Sonnenschein said that in air or other gases he believed sound waves were pressure waves and could easily be measured, but in fluids there could be very little compression. On the other hand, transmission of sound is least rapid in air, better in fluids and best in solids, but that referred only to the sound produced in that medium. If the sound passes from one medium to another this does not obtain. If one produces a loud sound near a body of water the sound will not be transmitted rapidly or well. Perhaps this had some bearing on the fact that when going from the air into the fluid of the labyrinth there is a change from one medium to another, and it requires the action of the stapes in this transfer.

Dr. Holinger said that the passage of sound from air to water or from a solid body to water was a difficult question. He made this experiment many years ago while in swimming. He lay on the bottom of the lake in four feet of water while his friends made all kinds of noises, even shooting a revolver. He heard nothing at all while under water. In hearing by bone conduction the transmission of sound waves from the walls of the labyrinth to the fluid of the labyrinth certainly does not change the pressure in the fluid.

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